BENIGN, ENCAPSULATED TUMORS IN THE LATERAL VENTRICLES OF THE BRAIN

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DIAGNOSIS AND TREATMENT

BY

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CONTENTS

PREFACE vii

CHAPTER I

INTRODUCTION 1

CHAPTER II

SMALL PRIMARY TUMORS IN THE LATERAL VENTRICLES 8

CHAPTER III

INVASIVE AND MALIGNANT TUMORS OF THE LATERAL VENTRICLES 28

CHAPTER IV

THE LARGER ENCAPSULATED TUMORS CAUSING SYMPTOMS 44

CHAPTER V

ANALYSIS OF SIGNS AND SYMPTOMS OF ALL CASES (THIS SERIES AND THE LITERATURE) 154

CHAPTER VI

LOCALIZATION OF TUMORS IN THE LATERAL VENTRICLE BY VENTRICULOGRAPHY ... 170

CHAPTER VII

OPERATIVE TREATMENT OF VENTRICULAR TUMORS 175

CHAPTER VIII

PATHOLOGY 179

CHAPTER IX

SUMMARY AND CONCLUSIONS 184

BIBLIOGRAPHY 187

PREFACE

It was not long after the dawn of antiseptic surgery, when the great Lister, who made not only brain surgery but all surgery possible, probed the frontal lobe with his index finger in search of a brain tumor but missed it by a scant half inch. Even had he reached the growth, the result could have been scarcely more than a spectacular feat. The soil must be intensively cultivated over a period of many years before surgery was prepared to attack this difficult problem with even a fair degree of success. A vast technical armamentarium, special methods of combatting haemorrhages, safe and efficient anaesthesia must be provided; the functions and absence of functions of various parts of the brain, the effects of the surgeon's trauma to the contiguous and remote parts of the brain, methods of avoiding the maximum and control of the irreducible minimum of this trauma, the character and resultant surgical bearing of the many types of brain tumor, and finally methods of precision in localizing brain tumors—all and much more must be learned from intensive studies over half a century before the operative treatment of tumors of the brain could be even relatively effective and secure.

In this warfare against brain tumors—the supreme test of brain surgery—two entirely different lines of attack were necessary, and both must be perfectly synchronized. First the exact location of the growths, only too frequently concealed in the vast silent areas of the brain, must be determined and second, adequate weapons of attack must be provided and mastered. As has been true in most branches of surgery, the operative accomplishments far outstripped and for many years awaited further progress on the diagnostic front. Since, by mechanical aids, it is now possible to diagnose and localize with precision all brain tumors causing signs or symptoms of

intracranial pressure, the remaining struggle is now one of technical skill.

In the following pages the surgical and diagnostic story of a group of tumors occupying the lateral ventricle of the brain is presented. These together with a quite similar group recently reported from the third ventricle, are from the last secret abodes of tumors in the brain. The results cover a period of over fifteen years and necessarily progressively reflect the important advances that have occurred on the operative front during this period, i.e., the electrocautery, avertin anaesthesia and continuous suction.

CHAPTER I

INTRODUCTION

From a practical standpoint tumors in the lateral ventricle may be divided into two groups: (1) those that are too small to give symptoms and are, therefore, merely pathological curiosities, (2) those causing signs and symptoms. The latter group may in turn be sub-divided into (1) those that are benign and encapsulated, and (2) those that are invasive or otherwise malignant. The arbitrary consideration in this communication of only the benign, encapsulated and symptom-producing tumors in the lateral ventricles is due to the fact that they alone offer a permanent cure by extirpation.

From the literature I have been able to find reports of only twenty-five potentially benign tumors that appear to have arisen within the lateral ventricles, and at the same time were capable either from size or position of producing symptoms. The actual number of reported tumors in the lateral ventricle has indeed been much greater but the additional ones have been rejected for consideration because they were too small to produce symptoms or because they show evidence of malignancy. The neurological signs and symptoms may indeed be not materially different from the far more numerous cerebral gliomata that protrude into and frequently fill much of a lateral ventricle, or from the papillomatous tumors of the chloroid plexus which at times may even appear encapsulated, but are nevertheless malignant. For such tumors surgical benefits are, with exceptions, only temporary.

As long as tumors of this kind appeared to be so rare, and the defects of clinical localization, the uncertainties and dangers of surgery so great, it was but natural that they would be of interest only to pathologists. Not one of these tumors from the literature has been diagnosed by neurological examination.

A glimmer of hope was aroused by the efforts of Jumentíe, of Challiol and of Jacarelli to find a clinical syndrome by which tumors in the lateral ventricle could be localized, but this hope was quickly doomed to disappointment. After reviewing all available cases, including their own after the necropsy findings had been disclosed, they were forced to conclude that a clinical entity does not exist. Jacarelli, however, thought it possible that ventriculography might be helpful.

From the series of fifteen cases which form the basis of this communication and from which the neurological signs and symptoms have been analyzed, the writer is also forced to conclude that neither objective findings nor subjective symptoms can lead to the diagnosis of tumors in the lateral ventricle. There are, as will be shown, reasons for *suspecting* such tumors, but to explore a lateral ventricle through a deep cortical incision only on suspicion is indefensible.

Ventriculography has entirely transformed this picture. By this method the diagnosis and localization of all ventricular tumors causing pressure can be made with such accuracy that they may, without exception, be disclosed at operation. Moreover, this precision in diagnosis is not only easy and certain, but is attainable without risk to life or function. And although the operative removal of such deeply situated tumors is necessarily serious, increasing experience has already greatly reduced the risk to life and function.

It will also be seen that primary benign tumors in the lateral ventricles are much more common than the reports from the literature would lead us to believe. They represent perhaps 1 per cent of all brain tumors — a frequency slightly less than encapsulated tumors in the third ventricle.

The first primary benign tumor in the lateral ventricle to be found at operation and completely removed was reported by the writer in 1920 (Case I of this series). The localization was made by ventriculography (1918) after two attempts to find the tumor had been unsuccessful. This happened to be the first brain tumor to be localized by this method. This patient is

still alive and well (15 years after operation). Additional cases have been briefly mentioned in Volume XII of Dean Lewis' *System of Surgery* (1933).

I have been able to find in the literature only one other tumor in a lateral ventricle disclosed and removed at operation. This tumor, reported by VanWagenen (1930), was diagnosed by ventricular puncture and was successfully extirpated from the posterior horn. Microscopically it was found to be made up of alveoli of epithelium like the choroid plexus. The child lived over two years, when death resulted from another cause, but post-mortem examination of the head disclosed no sign of recurrence and no metastases.

The encapsulated tumors in the lateral and third ventricles are most rewarding exceptions to the ill-founded general rule, long held in restraint over neurological surgeons, that "every tumor below the surface of the brain is a glioma." The wisdom of such a rule in the days before accurate localization of intracranial tumors cannot be questioned, but at the present time a far better dictum is "actually to see every brain tumor (there are occasional exceptions) and to know whether it can or cannot be removed." For there are now no tumors giving signs or symptoms that cannot be accurately diagnosed, precisely localized and disclosed at operation. Such a program means that all curable tumors, such as those of this group, those in the third ventricle, and many others, may be given the maximum opportunities afforded by surgical treatment—which offers the sole prospect of cure or even transient benefit.

Although we are chiefly concerned with the encapsulated tumors, it is perhaps advisable to give brief consideration to the other two groups, one of which (non-symptom producing) has been considered by some to be of similar origin and, therefore, potentially an operable tumor at a later time; the other—invasive tumors—gives signs and symptoms that are all too frequently indistinguishable from those of the encapsulated type.

SMALL BENIGN TUMORS NOT CAUSING SYMPTOMS

Much of the literature upon tumors in the lateral ventricle is concerned with this group of pathological curios. In an excellent treatise in 1886, Audry collected from the literature and classified all the tumors of the choroid plexus and cerebral ventricles; most of these were in the symptomless group. He divided the tumors into two groups: (1) exaggerated development of the normal, and (2) those due to a foreign element (enchondroma, osteoma, etc.). In his collection are epithelial nodules by Chambard, Cornil and Ranvier; excrescences of connective tissue by Guérard, Demange, Dancereaux (called "fungeus" and "vascular" tumors); lipomas by Obersteiner, Wallmann, Haeckel; myxomas by Perreton; psammomas by Boscredon and Beigel; indurative hyperplasia of the glomus by Wenzel; degenerative cysts of the plexus by Bergeon. Although most of the above tumors are in the fourth and third ventricles, identical tumors obtain in the plexus of the lateral ventricle. It is interesting, in passing, that Audry makes mention of the fact that Wenzel first applied the name "glomus" to this well known enlargement of the plexus.

Wallmann's lipoma (1858) was the size of a bean, firm and lobulated; there was also a smaller one of similar character in the choroid plexus of the other lateral ventricle. Although a lipoma of the brain had been described by Meckel in 1818, Wallmann's case was the first of the choroid plexus. In addition to these lipomata, his patient had a colloid cyst of the third ventricle—also the earliest, I think, on record. Rokitansky is said by Wallmann to have reported (I cannot find the record) a bean-sized lipoma in the ependymal lining of the lateral ventricle and a second one on the inner side of the dura of the same case. Obersteiner's lipoma (1883) was 1.5 cm. in diameter and was adherent to the left choroid plexus.

Saxer (1902) reported a cherry-sized encapsulated tumor along the floor of the body of the lateral ventricle; it was made up of fibrous tissue and covered by ependyma, which in places dipped into the tumor; it also contained numerous calcareous

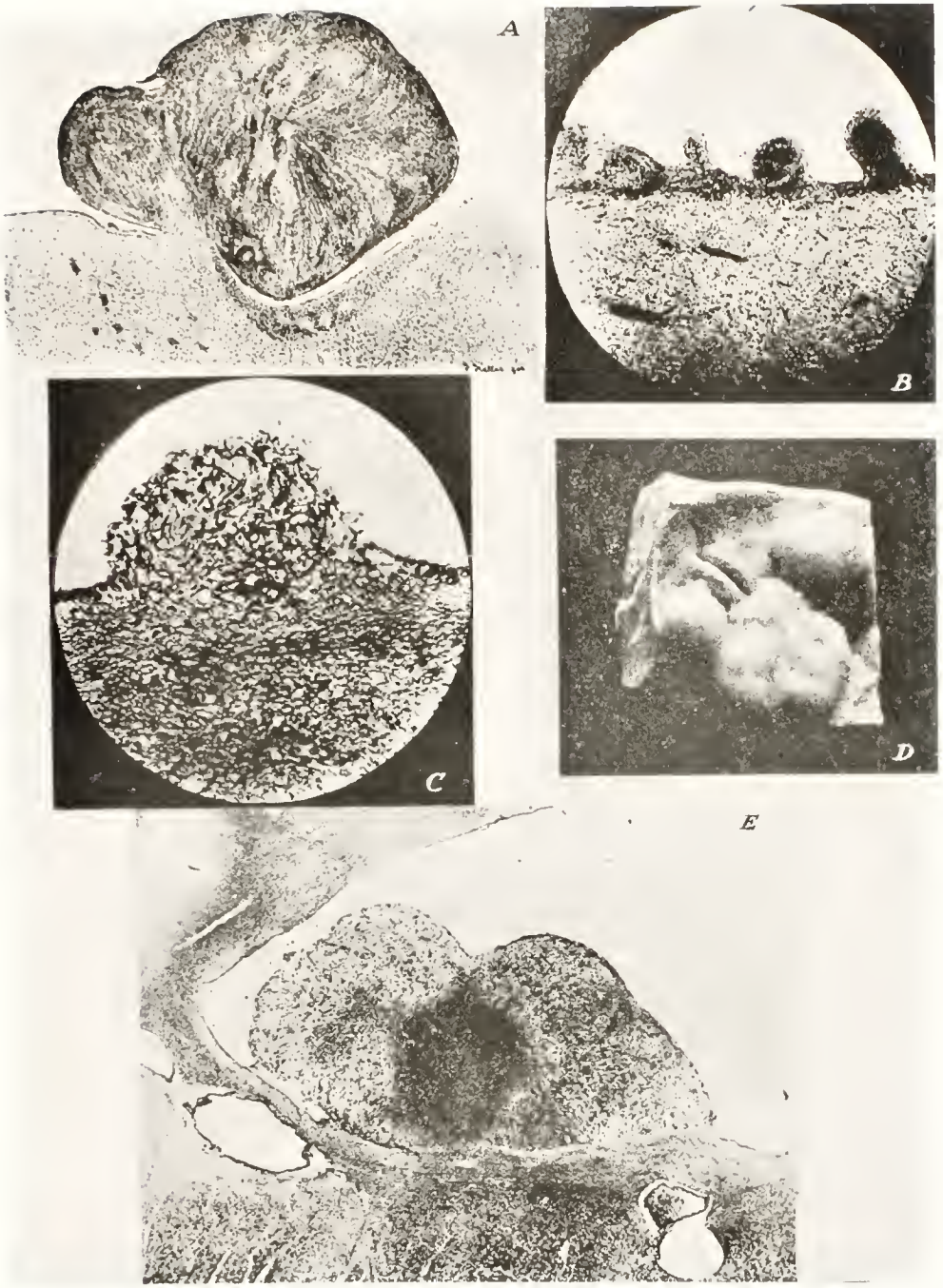

FIG. 1. *A*. VonWiller's tumor attached to the roof of the floor of the ventricle by a very small pedicle: the tumor is magnified.

B. Multiple tiny nodules projecting from the wall of the lateral ventricle in case reported by Margulis.

C. Glial fibers contained in the little tumors shown in Figure 1, *B* (Margulis).

D. Gross appearance of slightly larger multiple ependymal tumors in case reported by Rizzo.

E. Rizzo's tumor in section and magnified.

concretions. Vonwiller's tumor (Fig. 1, *A*) is perhaps also one of similar type. The small tumor reported by Claude and Loyez, and causing symptoms, is not unlike Vonwiller's. Indeed these writers regard the tumor as an example of granular ependymitis.

In this connection it is probably well to consider a group of small multiple fibrous excrescences that are not infrequently found along the ventricular walls. Margulis (1913) describes and gives illustrations of five such cases—all incidental findings at necropsy—and calls them examples of granular ependymitis (Figs. 1, *B* and *C*). They may be in great numbers throughout the ventricular system, of varying size, and may or may not be, although usually they are, covered by ependyma. They may be attached by a small pedicle, but are usually hillocks with a broad base. They are made up of proliferations of subependymal glia. Although innocent looking, and doubtless actually so, they too may be, as their seemingly similar macroscopic structure indicates, like Saxer's isolated tumor and perhaps a forerunner of the larger benign ependymal gliomata. A splendid thesis on this so-called "ependymitis" is presented by Merle (1910). He considers this process as also the probable cause of the cicatricial stenoses of the aqueduct of Sylvius which is so frequently responsible for hydrocephalus at all periods of life. He gives Brenner (1694) credit for the first description of this lesion which in the first half of the nineteenth century received the attention of Andrae (1820), Bayle (1826), and Rokitansky (1844), the latter reporting five cases. More recently cases of *multiple* subependymal gliomatous nodules have been reported by Rizzo (1926) (Fig. 1, *D* and *E*) and Jumentié and Barbeau (1926).

Cornil (1901) described a raspberry-sized tumor, 3 cm. in greatest diameter, encapsulated and containing several small cysts, but lying in the white matter of the brain and attached to the *pia* by a small vascular tract. It was made up of villi of epithelium like "choroid plexus or ependyma" and "was probably a rest." Since the specimen was obtained at necropsy,

it can be fairly safely assumed that the tumor was not a metastasis from a primary tumor in the choroid plexus. One might on first impression be incredulous of a tumor of this origin at a distance from the lateral ventricle, but in the case of Davis and Cushing, from which a small fragment was taken for diagnosis, the tumor was entirely without the ventricle. That small tumors of this type (Cornil's case) account for the subsequent growth of the larger tumors causing symptoms (Davis and Cushing's case and our Case X) appears to have a greater degree of evidence than in the preceding type of ependymal gliomata. Moreover, the epithelial tumors arising at a distance from the ependyma could only be explained by "rests."

Abercrombie (1835) reported a small tumor of hazel-nut size attached to the choroid plexus of each lateral ventricle. The absence of more detailed information, especially microscopic studies, prevents its classification with any degree of certainty, but it is probably of the type shown in our appended Cases IV, V, VI, and VII.

CHAPTER II

SMALL PRIMARY TUMORS IN THE LATERAL VENTRICLES

A. SMALL SOLID TUMORS

In recent years seven cases with nine small non-symptom producing solid tumors of the choroid plexus have been disclosed in my operative and post-mortem material. These were exclusive of the numerous cysts and psammomata, all of which may be regarded as normal findings. All of these tumors were situated in the glomus of the plexus, Fig. 2. In two cases bilaterally symmetrical tumors of identical morphology were present in the same individual. Gross and microscopic pathological notes on these tumors are as follows:

Case I

Necropsy specimen: P. No. 9919. From an infant with advanced hydrocephalus (Fig. 2).

Gross. Irregularly rounded hard mass, size of hazel nut, is firmly attached to the ependymal lining of the optic thalamus and is apparently a bud from the brain and not a primary tumor of the plexus. The choroid plexus can be seen entering and terminating in the mass. There is no posterior continuation of the plexus in the greatly malformed brain.

Microscopic (*Fig. 2A*). Mass is only partly surrounded by ependymal epithelium. In one place brain tissue grows into the tumor which is made up largely of fairly compact fibrous tissue closely resembling subependymal glial tissue. A few immature alveoli like those of the choroid plexus are engulfed at the base of the tumor, i.e., where it springs from the brain.

In a second and similar nodule lying alongside, the fibrous element is less compact and more like areolar tissue. In a section stained for glia, no glial fibers are present.

There is no iron pigment.

Diagnosis. Ependymal fibroma.

Case II

Necropsy specimen: P. No. 12046. From an infant with advanced hydrocephalus.

Gross. Oval shaped, reddish-black tumor, size of an almond, dangles from the glomus of *each* choroid plexus like earrings.

FIG. 2. Case I. P. No. 9919. Tiny fibrous nodule in glomus of choroid plexus

The tumors are symmetrical. Tumors are incidental findings and can have no possible bearing upon the production of hydrocephalus.

Microscopic (*Fig. 3*). The mass of the tumor is made up of an embryonal type of tissue with thin filmy processes and scant nuclei that stain poorly. Throughout the tumor, but especially near the periphery, are numerous epithelial lined alveoli of varying size. Clearly these are metamorphosed alveoli of the choroid plexus, but most of the alveoli are of

tremendous size when compared to the normal and the epithelial covering is made up of flat cells which in many places are reduced almost to the thinness of endothelium, but here and there cubical cells still persist. The interior of the alveoli is made up of a loose areolar tissue, not greatly different from

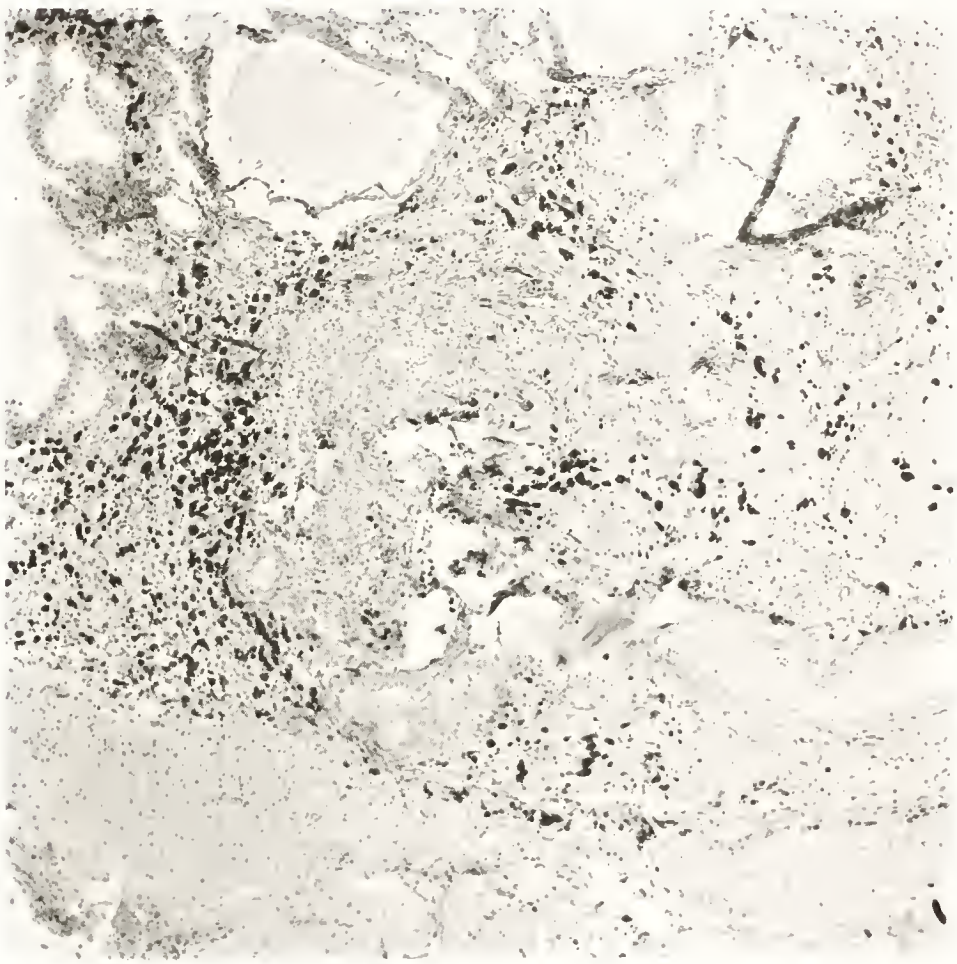

FIG. 2.1. Photomicrograph of Case I. In the upper left-hand corner is some epithelium of choroid plexus. The small black masses are iron pigment.

the tissue described above as forming the body of the tumor. Usually there is the central capillary or larger vessel so characteristically placed in the choroidal alveoli. In places a low cubical epithelium covers the surface of the tumor, but in most places this is absent and only somewhat more compact con-

nective tissue forms the lining. In other places the abnormal alveoli reach the surface and form its only covering.

No calcareous bodies are seen in the tumor. Normal choroid plexus lies at the base of the tumor and the tumor is continuous from its tissue. There is much iron pigment throughout the tumor.

Diagnosis. Adenoma of the choroid plexus.

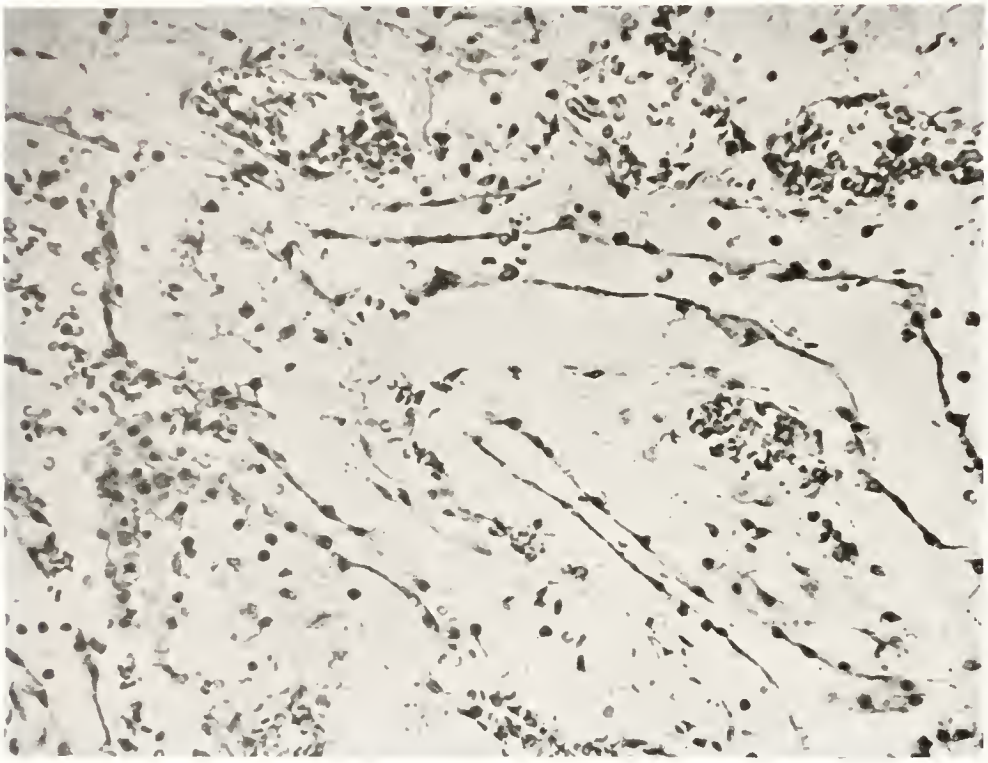

FIG. 3. Photomicrograph of tumor in Case II, P. No. 12046, showing papillae of choroid plexus with greatly modified flattened epithelial covering.

Case III

Necropsy specimen: P. No. 12289. From a woman aged 59, with a dural endothelioma under the left temporal lobe.

Gross. Small, firm, round tumor, measuring 7 mm. in diameter, in glomus of the right choroid plexus. On section one can see tiny cavities.

Microscopic (Figs. 4A and 4B). Tumor is lined in most

places by single layer of cubical epithelium like that of choroid plexus. This epithelial layer can be traced into the epithelium of the adjacent plexus. Beneath is a layer of fairly compact connective tissue, within which is an incomplete ring of numerous circular psammoma bodies. Some of the psammoma

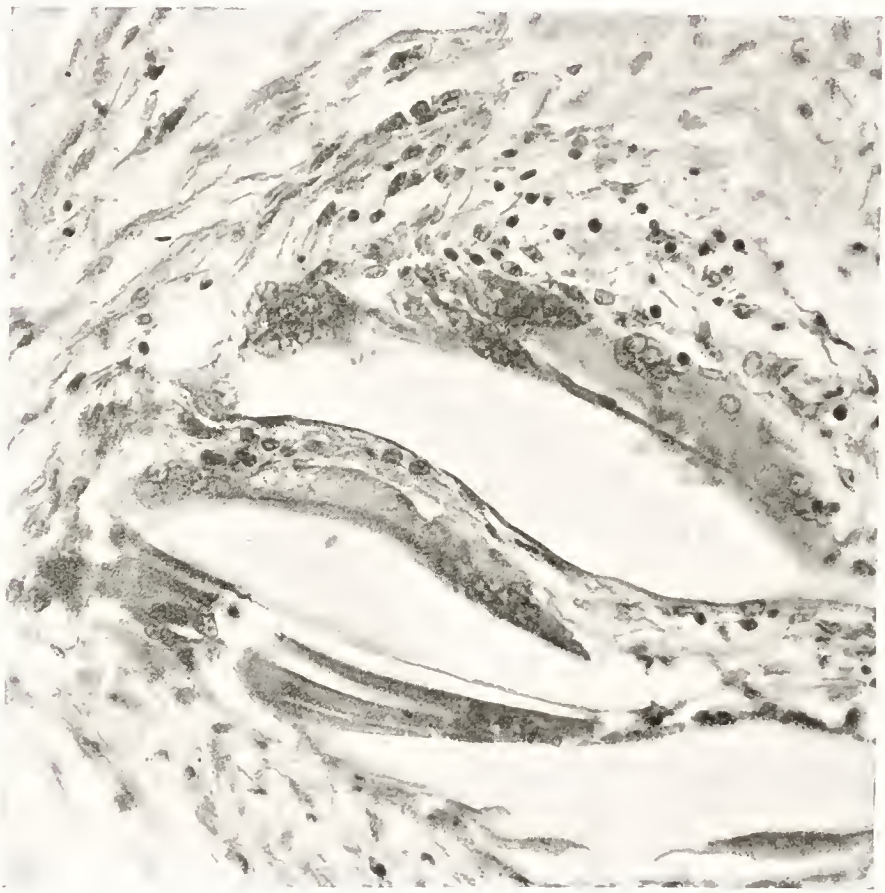

FIG. 4A. Case III, P. No. 12289. Showing cysts with projecting masses of large cells into lumen.

bodies are also located in the deeper part of the tumor, others are in the adjacent choroid plexus. The body of the tumor is made up of fairly compact connective tissue with few nuclei. In a few patches a looser areolar tissue predominates.

The striking feature of the growth is the series of minute cysts just visible to the naked eye and located in the center of

the tumor. Lining these and projecting into them as numerous spurs are masses of large cells suggesting metamorphosed epithelium. In places there are dozens of large round vesicular nuclei clustered together in masses of deeply red-stained, undifferentiated protoplasm. Perhaps such areas may be looked upon as giant cells, but the absence of cell boundaries in many places makes it difficult to classify them as cells. They

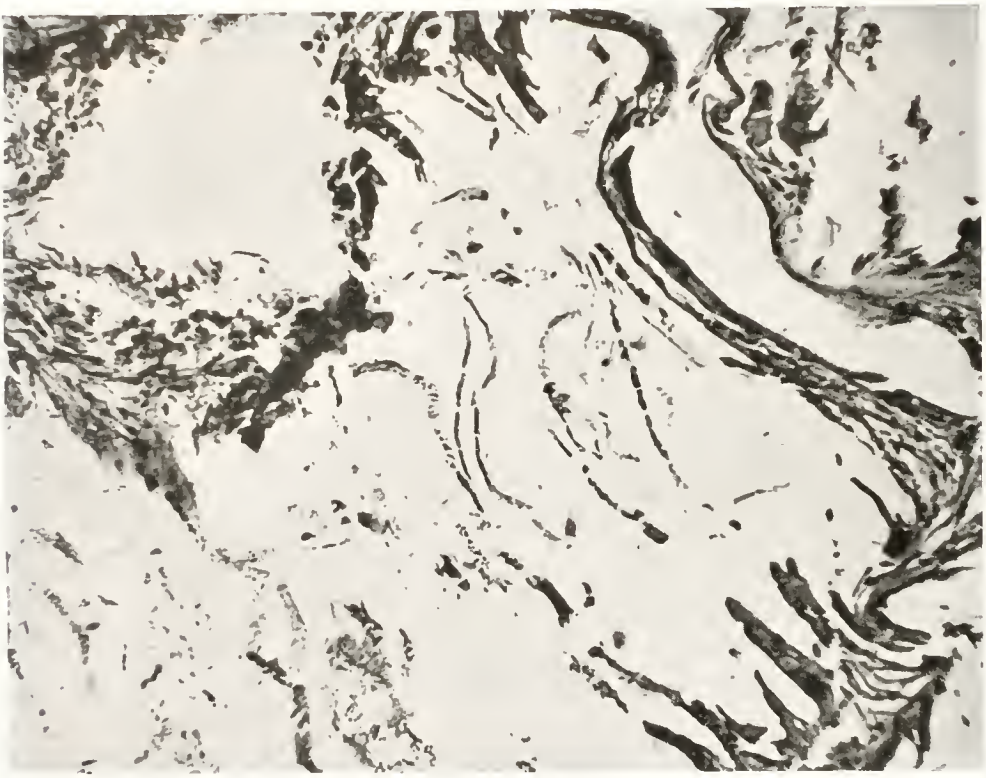

FIG. 4*B*. Case III. High power photomicrograph showing giant cell formation. Spaces are due to crystal formation.

rather suggest isolated patches of syncytium. Lining all the cysts, and doubtless, therefore, the cause of them, are such bizarre collections of epithelial (?) material. Throughout the cysts are necrotic pink-staining strands which are doubtless exfoliations of the projecting spurs.

These epithelial masses suggest the origin of some tumors of the third ventricle described in a recent article on that subject.

Iron pigment is scattered throughout the tumor.

Diagnosis. Perhaps tumor of embryonal cells from which epithelium of choroid plexus or ependyma is derived.

FIG. 5. Case IV, P. No. 9718. Photomicrograph showing loose reticular connective tissue forming the tumor.

Case IV

Necropsy specimen: P. No. 9718. From a man aged 54 with a glioma of the temporal lobe.

Gross. Pea sized, firm, sharply defined, round tumor in the glomus of the left choroid plexus.

Microscopic. It is lined externally by ependymal epithelium.

The interior is made up of a reticular tissue with scant nuclei or almost none in places (Fig. 5). Here the numerous strands of pink-staining tissue, quite compactly arranged, coalesce into a homogeneous pink-staining material (Fig. 6). In some places there are small dilated spaces with thin trabeculae;

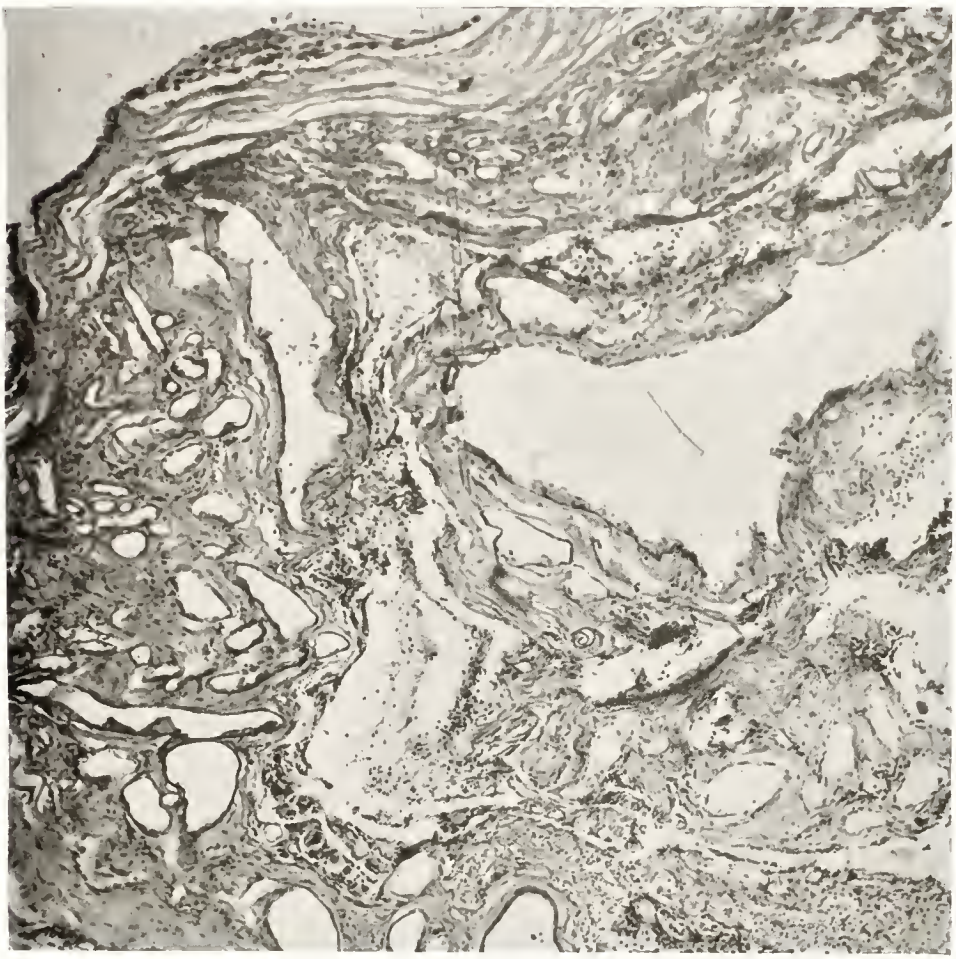

Fig. 6. Case IV, p. 9718. Photomicrograph showing more dense fibrous tissue with many small cystic cavities.

much the same microscopic appearance obtains as the interior of a cyst of the plexus, though on a very reduced scale and only in small patches. In other places the reticular tissue contains many scattered nuclei. These definitely more viable parts contain occasional small blood vessels.

In places there are myriads of tiny circular calcareous areas which frequently coalesce into masses of varying size and are exactly like similar areas so frequently seen in degenerative cerebral tissues and tumors. In addition there are numerous larger characteristic laminated spherical psammoma bodies of fairly uniform size so commonly present in the choroid plexus.

No iron pigment can be found.

Diagnosis. Fibroma of choroid plexus.

Case V

Necropsy specimen: P. No. 10981. From an infant with advanced hydrocephalus.

Gross. Small, firm, round, reddish-brown tumor, size of a hazel nut in the glomus of the right choroid plexus.

Microscopic. The tumor is lined externally by a layer of loose areolar tissue—no epithelial covering can be seen. The outer layer is filled with pigment throughout. Beneath is a layer of fibrous tissue with few nuclei.

The body of the tumor is made up of loose connective tissue and at other places of more compact strands. There are numerous cystic spaces of varying size, one covering almost one-eighth of the cross section of the tumor. These spaces are lines by endothelium for the most part, but in places there is a low epithelium suggesting ependyma or flattened choroid plexus. Numerous calcareous concretions are collected near the center and much pigment is scattered throughout the tumor.

Diagnosis. Fibroma of choroid plexus.

Case VI

Operative specimen: P. No. 47561. From an infant with advanced hydrocephalus (Fig. 7.1).

Gross. Hazel-nut sized, round, firm, reddish-brown tumor in the glomus of the choroid plexus. It was removed when both choroid plexuses were extirpated.

Microscopic (*Fig. 7B*). The nodule is in large part made up of fibrous tissue containing few nuclei. There are many vessels of relatively large size; some are lined by a much thickened intima, but other coats are missing, although the fibrous tissue tends to be arranged concentrically around the vessels and joins with other similar strands to form the bulk of the tumor. Pigment is abundant throughout the nodule. The tumor

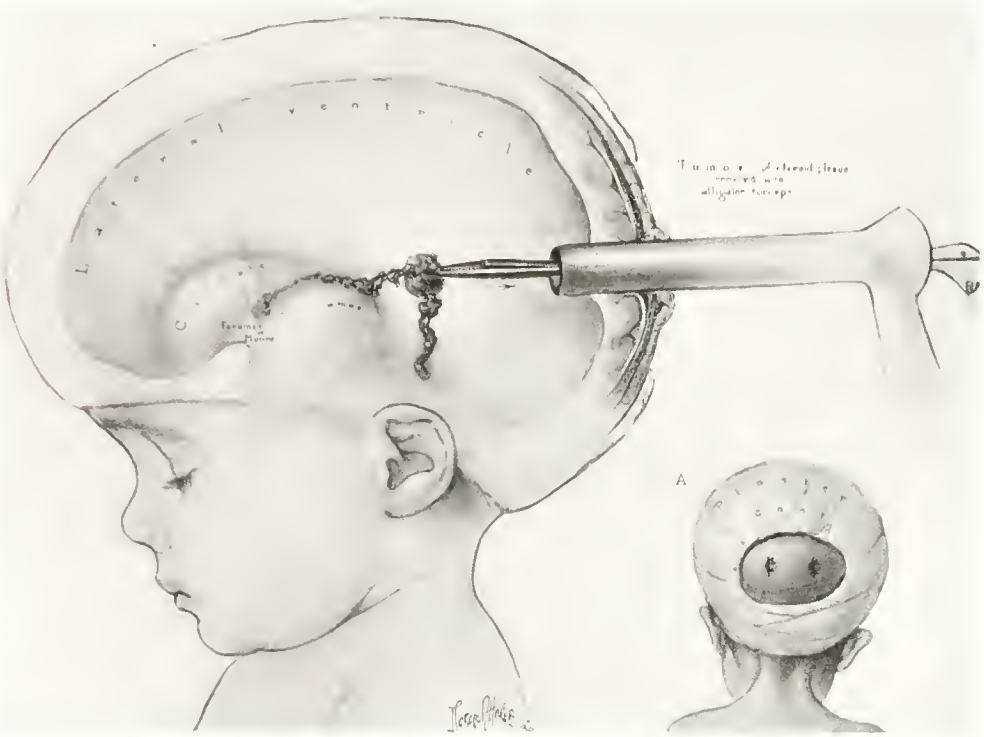

FIG. 7.1. Drawing of tumor Case VI, P. No. 47561. Tumor was removed through the ventriculoscope. The choroid plexus was extirpated from each lateral ventricle in the treatment for communicating hydrocephalus.

grows from the choroid plexus, some of the alveoli of which are incorporated where the tumor lies adjacent.

Diagnosis. Fibroma of choroid plexus.

Case VII

Operative specimen: P. No. 54235. From an infant with advanced hydrocephalus. In exactly the same location in

the glomus of *each* choroid plexus was a small round firm hazelnut sized tumor. Both were removed at operation with the choroid plexuses. The gross appearance and microscopic picture were exactly like P. No. 47561.

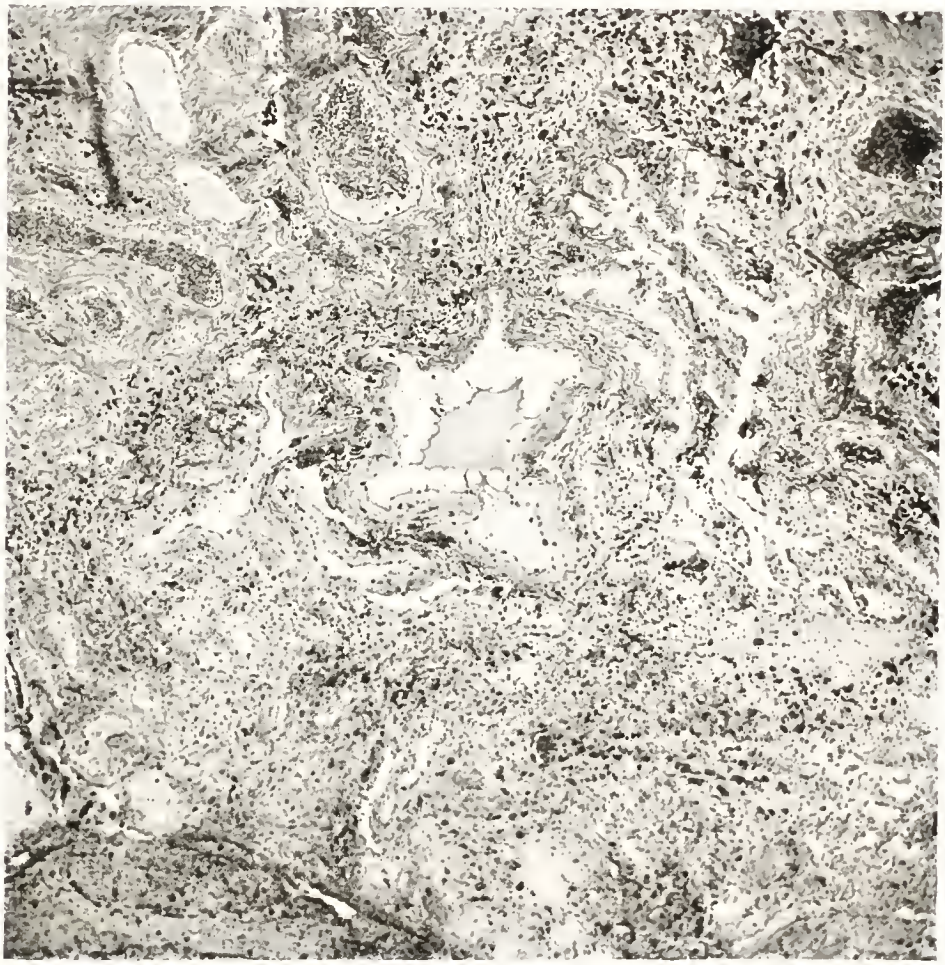

FIG. 7*B*. Photomicrograph Case VI, P. No. 47561. The tumor was made up of rather compact connective tissue. The dark dots throughout the section are deposits of iron pigment.

Diagnosis. Fibromata of choroid plexus.

Among these nine tumors (7 cases) there are, as the appended diagnoses indicate, at least four distinct types. The gross and microscopic appearances of case I are clearly those of an ependymal fibroma in miniature. This tumor is probably like those

of Saxer, Vouwiller and Claude and Loyez, and probably also of Margulis and Merle and others. There can scarcely be a doubt in our case, in which the brain was a series of gross malformations, that the tumor was a misplaced "rest."

Case II must be reported as an adenoma of the choroid plexus. Although the alveoli are far removed from the adult type, they are nevertheless unmistakably of this origin. Even so, the greater part of the tumor is made up of an uninterrupted embryonal connective tissue of areolar type, which is quite similar to that forming the body of the papillary formations that are lined by the metamorphosed choroidal epithelium. It is interesting that this tumor is bilateral and symmetrical.

Case III is unlike any in the literature. Its bizarre, syncytial-like masses with a tendency to giant formations resembles more closely some of the large benign tumors of the third and lateral ventricles than any other formation that comes to mind.

Cases IV, V, VI and VII, although differing somewhat in the texture of the connective tissue which makes up the tumors' mass, are probably of similar type. Possibly they are like the "exereseences" of connective tissue of Chambard, Cornil and Ranvier, Guérard, Demange, Daneereaux, etc.

The presence of so much iron pigment in all of these tumors is of interest and perhaps of significance, although its interpretation is not clear. One would naturally think of a hemorrhage with subsequent organization as a cause. Against this theory is a similar diffuse pigmentary deposit in Case II which is unquestionably a true neoplasm. Against the explanation of an organized hematoma are the bilateral, symmetrical tumors in Case VII. That hemorrhages should occur in the exact same spot in the choroid plexus in so many instances is scarcely conceivable; and finally, we are unacquainted with the existence of hemorrhages into the glomus of the choroid plexus as the result of birth trauma or other causes. It is my impression that they are actual benign tumors arising in the connective tissue of the choroid plexus. I have no explanation for the presence of such large amounts of iron pigment.

It is not difficult to believe, but is impossible to prove, that these small tumors of different types are forerunners of the larger symptom-producing tumors of later years.

It will also be observed that six of the nine tumors (5 of 7 cases) were from hydrocephalic infants; although the hydrocephalic specimens are both actually and relatively fewer than those of other types of pathological material. The early age of their appearance as well as the histological structure in certain cases (I, II and III) suggests the origin of tumor from congenital anlage or "rests." Perhaps the greater incidence among hydrocephalic children in whom congenital malformations are unquestionably much more common, may further suggest but does not prove the origin of tumors from congenital "rests."

B. CYSTS OF THE CHOROID PLEXUS

Cysts of the choroid plexus have long received the attention of pathologists because of their striking appearance, as well as their frequency. Schnopfhagen (1876) and Audry (1886) were particularly interested, describing them as "cystic degeneration." Curiously the choroidal cysts are rarely seen in the plexuses of the third or fourth ventricles, but are exceedingly common in the lateral ventricles, particularly during adult life. They may be single or multiple; may be located at any part of the choroid plexus, but the glomus is unquestionably the favored seat (Fig. 8). Usually when cysts are found in one lateral ventricle they will be present in the other also. They have a thin transparent wall and contain a clear watery fluid.

The microscopic appearance of cysts of the choroid plexus is given in the following two cases:

Case I

Path. No. 1293. Adult male. Multiple cysts of both choroid plexuses (Fig. 8A).

A very thin outer layer of flat epithelium—probably ependymal—lines the surface of the tumor. Cyst itself is criss-

crossed with delicate strands with occasional flat nuclei which are wider than the trabeculae and cause a bulge. Numerous calcareous bodies. One gets the impression that the cysts develop in the areolar connective tissue spaces and not in the choroidal alveoli.

Case II

Path. No. 47337. Adult male. Single large cyst in the glomus of the choroid plexus.

FIG. 8. Typical instance of multiple small cysts of the choroid plexus. Specimen removed at necropsy.

A thin layer of fibrous tissue with very few nuclei lines the surface. No sign of an epithelial lining of any kind is visible. The interior is a network of very delicate strands so thin that the occasional nuclei cause a distinct bulging to either side. In many places the strands have broken and coalesced forming a slightly pink (eosin) staining degenerative debris either without nuclei or with scant nuclear remains. The compartments within the cyst suggests an exaggeration of the normal tissue spaces within the choroid plexus. Choroid plexus containing numerous psammoma bodies are attached to the cyst.

From their microscopic structure one is led to believe that

cysts of the choroid plexus are not of epithelial derivation, for an epithelial layer is everywhere lacking. The web-like character of the interior, together with the retention of a striking resemblance to the loose connective tissue and spaces of the normal choroid plexus, suggests that they are cysts arising

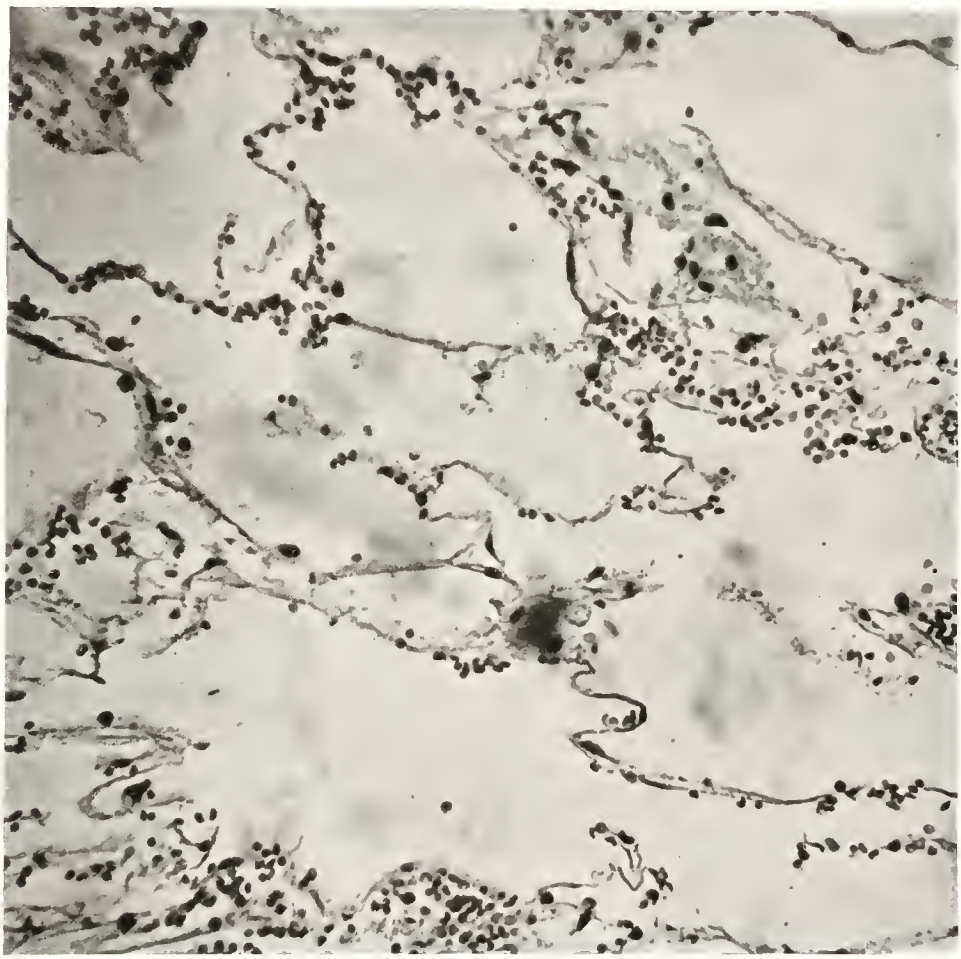

FIG. 8.1. Photomicrograph showing microscopic appearance of one of the small cysts shown in Figure 8.

from the connective tissue—admittedly an unusual assumption. If we knew that there were lymphatics in the choroid plexus, these compartments might be looked upon as dilated lymphatics.

Cysts of the choroid plexus have no resemblance, either gross

or microscopic, to the colloid (epithelial) cysts that, so far at least, have been found only in the third ventricle.

I know of no instance where a cyst of the choroid plexus has caused symptoms. Whether they may be related to the huge cysts of Cayley and Brown, Jimson and Denet, and perhaps of Lydston, is, of course, open to question, but the absence of histological data in the above cases makes such speculation idle. While it is by no means improbable that cysts of the

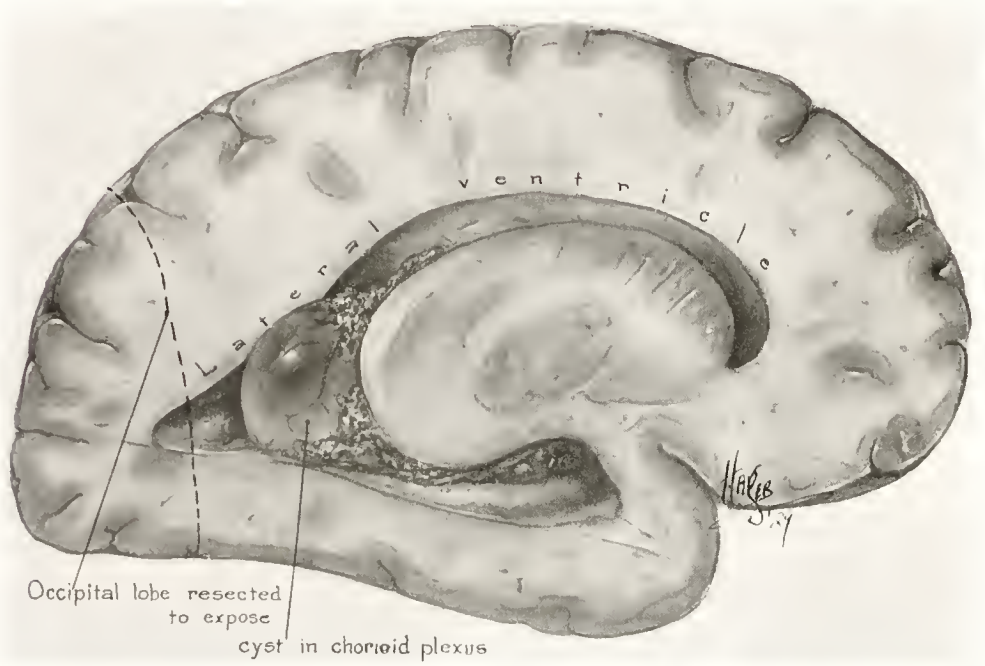

FIG. 9. Single large cyst in the glomus of the Choroid plexus; though it filled the ventricle it had caused no symptoms.

choroid plexus may at a later date be found to cause obstruction to the ventricular system, the great frequency of cysts without any evidence as yet of one causing symptoms is proof that such an outcome will, at least, be very rare. However, on one occasion I was misled into removing a large cyst of the choroid plexus, though it later proved to be a perfectly innocent lesion (Fig. 9). Ventriculography had beautifully disclosed a cyst at the glomus of the right lateral ventricle exactly filling this

cavity, but causing no hydrocephalus in the temporal horn, as it would necessarily do if actually obstructing the ventricle. An actual tumor was, therefore, excluded by ventriculography, but when, a week later, the patient suddenly became comatose, being so apprehensive that a sudden occlusion by the cyst

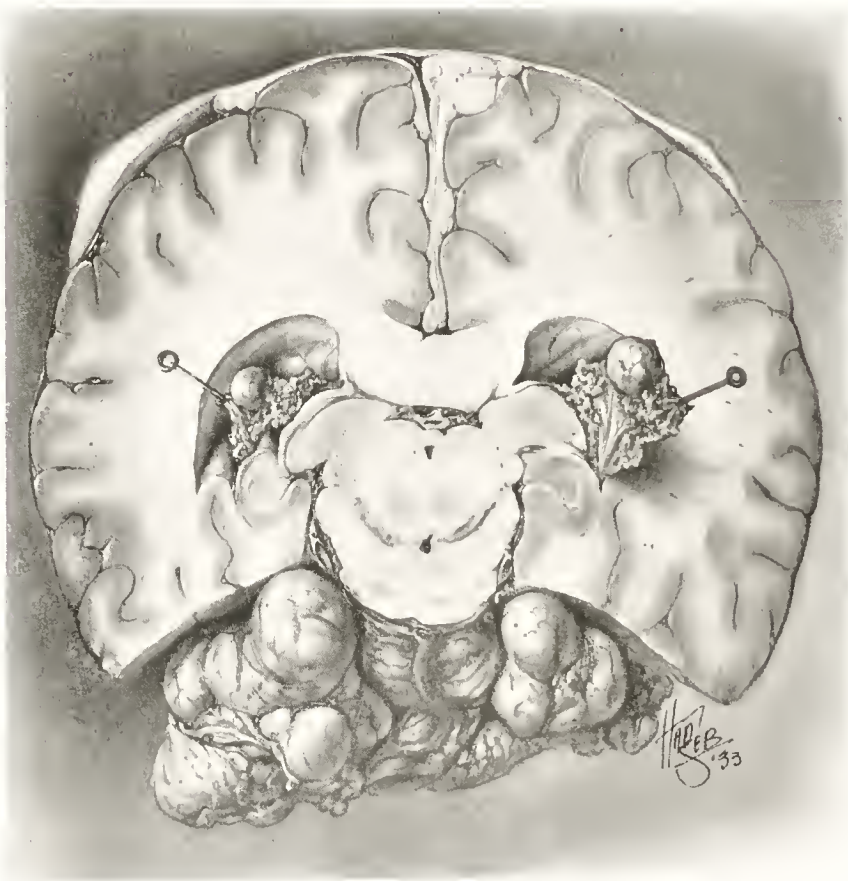

FIG. 10. Necropsy specimen showing psammoma in the glomus of each lateral ventricle.

might have occurred, in the absence of other evidence, I removed the cyst. Necropsy disclosed an intracerebral hemorrhage from a tiny aneurysm on the anterior cerebral artery.

C. PSAMMOMATA

Psammomata or small concretions in the choroid plexus of the lateral ventricles, especially in the glomus, long ago engaged

the interest of Boseredon (1855) and Biegel (1869). There are few choroid plexuses of adults that do not show psammoma bodies. When assembled in sufficient number they form palpable concretions and are frequently visible in x-ray of the head—perhaps 5 per cent of all late adult x-rays and occasionally before the twenty-fifth year. They are, however,

FIG. 104. Area of calcification of psammoma. Inset shows x-ray of psammoma taken after the choroid plexus had been removed from the brain.

but secondary changes in the life history of the choroid plexus. Similar isolated psammoma bodies occur in other meningeal tumors, particularly dural endotheliomata, and at times in such great numbers that the term psammoma has been applied to them. Psammoma bodies are also present in many of the tumors, both small and large, that arise within the lateral ventricles. In most of the tumors reported in this article, regardless of their character, psammoma bodies have been

found. Psammomata of the choroid plexus probably carry no significance and so far as I know never give rise to symptoms of any kind. Clinically they must not be confused with calcified deposits in tumors and even elsewhere in the brain. Areas of calcification (not the psammomata type) are indeed quite common throughout the cerebral hemispheres and when so situated are one of the causes of epilepsy.

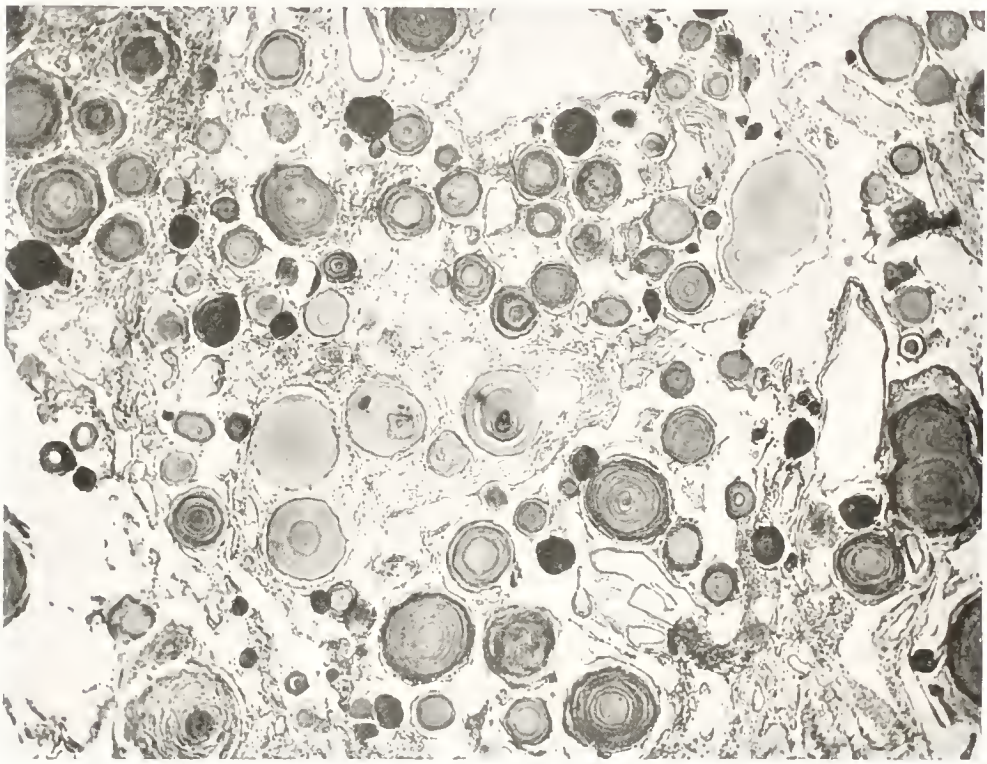

FIG. 10*B*. Photomicrograph showing typical microscopic appearance of the psammoma.

In one of the writer's cases of Recklinghausen's disease with numerous intracranial tumors of varying size, the x-ray showed an unusually large calcification in each choroid plexus (Fig. 10.1). At necropsy each glomus contained an extremely hard nodule about as large as a hazel-nut (Fig. 10). X-ray of these structures removed from the brain duplicated the original shadows (inset Fig. 10.1). Microscopically the

tumors were made up of myriads of psammoma bodies (Fig. 10*B*). On the interior of one tumor was a mass of compact fibrous tissue, such as is frequently seen in the choroid plexus but of greater size. It was not, however, at all like the Recklinghausen's tumors, to which it was, therefore, unrelated.

CHAPTER III

INVASIVE AND MALIGNANT TUMORS OF THE LATERAL VENTRICLES

A hard and fast line of demarcation between benign and malignant tumors within the lateral ventricles is occasionally impossible until the ultimate test of time has been applied. In most instances the gross appearances of the tumor—encapsulation and lack of invasion—are adequate, but there are abundant exceptions, particularly among the tumors of the choroid plexus which may be perfectly encapsulated and yet inherently malignant. On the other hand, the ependymal fibromata or gliomata are not entirely encapsulated for they spring from the subependymal glial tissue and yet they are enucleable and time has proven them to be curable. Except for the point of attachment—it is both small and superficial—of the ependymal gliomata, the tumor has every gross appearance of benignity.

In determining the benign or malignant character of ventricular tumors microscopic studies, unassisted, are even more unreliable. Certainly Bailey's attempts to differentiate ependymal gliomata by cytological features are unsound and misleading. Among his ependymal gliomata are grouped both malignant and benign tumors, so that from the practical point of view of a surgeon one gets nowhere. It is far better to apply the term "ependymal glioma" to a tumor that by practical test is sharply differentiated from all other gliomata in the brain.

Microscopic diagnosis in these as in other tumors attains increasing value only after correlation with the gross observations and especially with the ultimate results following extirpation. Neither the gross nor microscopic appearance of the tumor is as yet an infallible guide concerning the character of

the tumor and at times both together fail in the task, the life history of the growth being the final test.

Most of the intraventricular tumors reported in the literature have been immediately eliminated from consideration because they are merely extensions of large gliomata arising in the cerebral hemispheres. They are many times more frequent than the primary ependymal gliomata and fibromata and the

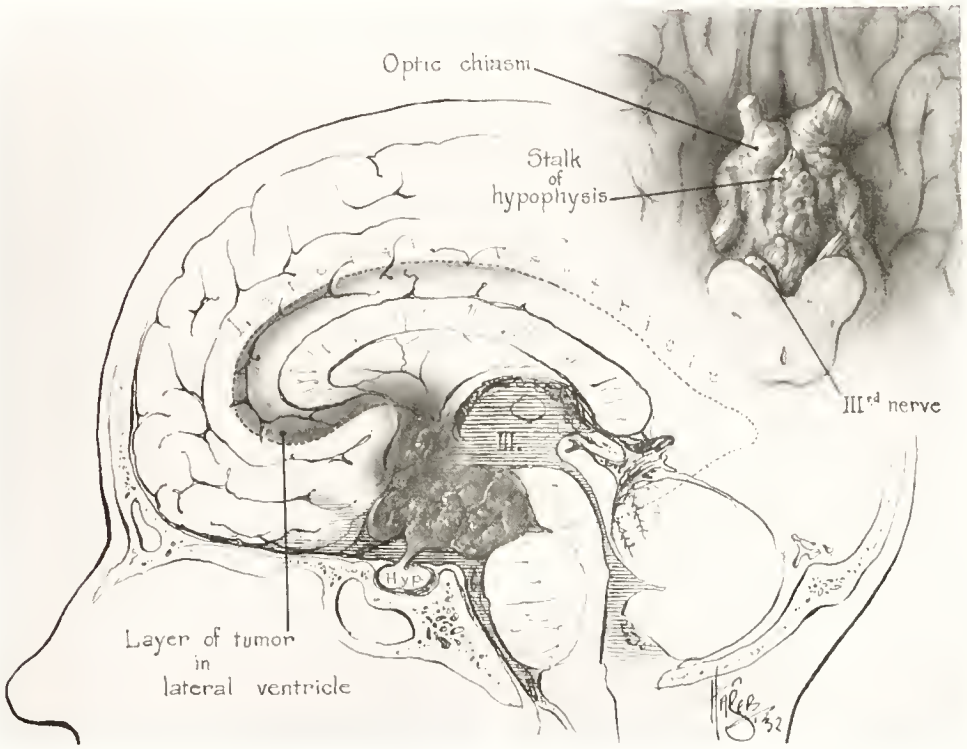

FIG. 11. Ependymal glioma in the lateral and third ventricle. It had grown through the foramen of Monro.

ventricular extension carries no diagnostic or therapeutic interest. In many reports it is impossible to tell either from the gross or microscopic description whether the intraventricular tumor belongs to the benign or malignant group; attempts at classification will, therefore, doubtless be attended by mistakes. Upon the macroscopic appearance I have excluded tumors reported by Wolff (1921), Challiol (1929) and Livierato and Cosmettatos (1929), all reported as ventricular tumors.

On the other hand, some doubt remains that other tumors included among the benign group actually belong there Jumientié, Wätzold, etc.

a. Malignant ependymal gliomata. That malignant, invasive gliomata may also arise from subependymal glial layer is also quite probable. At least there are gliomata that skim the surface of the ventricles and spread over a very extensive area, frequently passing through the intraventricular foramina into the third and the opposite lateral ventricle. The brain itself is but little involved. Figure 11 is a drawing of such a tumor from our experience. The patient had adiposity, somnolence, polyuria and later signs of intracranial pressure.

Prautois and Etienne (1894), Hirsch and Elliott (1925), and Natorek (1914) report tumors of this general character. In Prautois and Etienne's case the tumor extended through the aqueduct of Sylvius into the fourth ventricle and thence into the spinal canal. They call the tumor a primary sarcoma—a doubtful type of primary tumor in the brain.

b. Malignant tumors of the choroid plexus. The malignant tumors arising from the choroid plexus are interesting because their gross appearance may at times suggest a benign encapsulated growth and the histological pattern is that of the choroid plexus being not unlike a benign papilloma or adenoma. Many of these tumors appear in the early months or years of life and rapidly attain great size. Some indeed appear perfectly encapsulated, as in one of our cases, but usually they grow so luxuriantly that the surface erodes and all signs of encapsulation disappears. The base of the growth is usually so firmly and broadly attached to the brain tissue that removal without leaving remnants is scarcely possible. The tumors are very friable because of the high cellular and scant fibrous components. They are reddish-brown to red in color because of their vascularity. Some of them are malignant because of their strong tendency to local recurrence, others because they metastasize through the blood to all parts of the body, precisely like the adenocarcinomata which they resemble histologically.

In fact throughout the literature they are classified as adenocarcinoma or simply carcinoma. Some are doubtless spread by the cerebrospinal fluid for they give rise to independent nodules elsewhere in the ventricular system.

Just one hundred years ago Guérard reported perhaps the first case of a tumor of the choroid plexus. It was located in the posterior horn of a child of three years and was as large as a hen's egg. Although it was said to have produced no symptoms, the baby dying of measles, it is difficult to believe that a growth of this size was not the actual cause of death, especially since we now know that tumors of this type may long be silent and suddenly fulminate. The histological appearance of this tumor was described as being like that of the choroid plexus "from which it arose." No metastases were disclosed at necropsy, nor did the tumor appear to be invasive. It is entirely possible that it may have been a benign tumor, but in view of the rarity of benign tumors of the choroid plexus and the overwhelming tendency to malignancy, it appears impossible to regard it as benign. As far as one can judge from his description, it closely resembled my second case of malignant tumor of the choroid plexus.

LeBlanc (1866) reported an unquestioned malignant tumor of the choroid plexus. In addition to the primary growth in the lateral ventricle "as large as a child's fist," two small subpial transplants with identical microscopic appearance were found in the cerebral cortex. Bielchowsky and Unger (1906) described a much similar case. A tumor had been extirpated from a cerebral hemisphere and sometime later, at necropsy, another nodule with the same histological appearance was found in the cerebellum. Spät (1882) and Rokitansky reported similar cases and classified them as adenocarcinomata of the brain—a type of primary tumor otherwise unknown in the brain. Bielchowsky and Unger also regard them as primary carcinomata. In the following year (1907) Atlee of Lancaster, Pennsylvania, removed a seemingly encapsulated tumor from the right Rolandic area. A splendid temporary

result followed its enucleation for the patient, who was a physician, regained his motor and sensory functions. Histological examination revealed the characteristic duplication of the choroid plexus. Doubtless the tumor was a metastatic nodule because it was cortical and not intraventricular. Another interesting feature that especially engaged the attention of Dr. Mills, who reported the case with Atlee, was paralysis

FIG. 12. Example of a huge malignant tumor of the choroid plexus taken from article by Somerford.

of the vocal cord in the contralateral side. Dr. Mills collected many reports from the literature to prove that the paralysis was of central and not peripheral origin. We now know from extirpation of a cerebral hemisphere that paralysis of the vocal cord does not follow. Recently Dr. Atlee advised me that his patient died several months after operation from symptoms that were referred to the thorax. It was his belief that

the patient died as the result of metastases from the original cerebral tumor—probably an adenocarcinoma of the choroid plexus.

Noodt, Lehoczky, Somerford (Fig. 12), and many others have reported cases of presumably malignant tumors of the choroid plexus in the third, fourth and lateral ventricles of the brain. VanWagenen in a splendid article in which he reports two tumors of the choroid plexus (one probably benign) from the lateral ventricles, collects thirteen additional cases from the literature. Also included in his review are thirty-two cases of similar type arising within the third and fourth ventricles. The favorite abode appears to be in the fourth ventricle (70 per cent of the cases). From the gross appearance of VanWagenen's second case in cross section, one would almost unhesitatingly venture the opinion that the tumor was a large invasive glioma, a diagnosis which, of course, is denied by the microscopic picture. This gross deception is merely evidence of the many forms tumors of the choroid plexus may assume and the ultimate need for a histological diagnosis for the sake of accuracy, even though the prognosis is not altered. Although all tumors of the choroid plexus, heretofore reported, have apparently been malignant (VanWagenen's probably excepted), benign tumors do occur. One of these I recently reported among a series of benign tumors of the third ventricle, and another is included in this paper. There is ample proof of the benignity in both instances. The histological appearance of the benign tumors of the plexus is perhaps clear enough, but I am not so sure that the microscopic evidence is adequate without the test of time, i.e., permanency of cure following removal of the tumor. Just as the malignant tumors grow rapidly, so their recurrence is equally prompt. In one of my two malignant tumors of the choroid plexus I felt reasonably certain of a permanent cure following the extirpation. The tumor was seemingly well encapsulated. Although adherent in places to the ventricular walls, it could be shelled out of its bed apparently in its entirety. Only a knowledge of

the malignancy of such tumors made me skeptical of an ultimate cure. But six months later signs of recurrence appeared and a large invasive mass was disclosed at the former site. The second case (necropsy specimen) was obviously malignant for it was a huge vicious ulcerating mass filling a greatly distended ventricle, and though circumscribed it was firmly woven into the brain over a wide base. Were it located outside the brain one would immediately suspect a "sarcoma" from its gross appearance.

Case I

J. S., 20 months old male baby was referred by Dr. Joseph J. Friedman, of Brooklyn, New York, with the diagnosis of a brain tumor.

The patient entered the Johns Hopkins Hospital in apparently good condition, but died suddenly three and one-half hours later. He was seen by the resident only a few minutes before the sudden collapse appeared. It was reported by the nurse that he was pale, temperature 106.6°; pulse imperceptible and the respirations very rapid and irregular. While the patient was in this condition the resident called and at once introduced a lumbar puncture needle into the fronto-parietal suture, which was widely separated, and 90 cc. of slightly bloody fluid escaped under tremendous pressure. At this time—which was fifteen minutes after the child was observed to be in desperate condition—the temperature had reached 108.4°; the ventricular puncture had no effect, and in forty-five minutes the patient was dead; at that time the temperature had reached 111 degrees.

The present illness began six weeks ago when the child became fretful; weakness developed synchronously in the right arm and face, and two weeks later in the right leg. The loss of motor power in the right leg became progressively more severe, and at the time of admission to the hospital he was unable to move either the arm or leg. He had stopped talking; there had been infrequent vomiting spells; both eyes were crossed,

each pointing toward the nose. Involuntary urination had been noted from time to time. There were no actual convulsions, although some twitching had been observed at times in the right side of the face. The anterior fontanelle is said to have closed at eight months.

X-ray of the skull had been taken by Dr. Friedman; this showed marked separation of all the cranial bones and convolutional atrophy. A lumbar puncture had also been done by Dr. Friedman twenty-four hours before; the fluid was under tremendous pressure. At this time he injected 98 cc. of air into the spinal canal replacing 105 cc. of fluid which had been withdrawn.

On admission to the Johns Hopkins Hospital patient was found to be well nourished, very fretful and uncoöperative, but there was nothing to suggest apprehension about his immediate condition. Since he arrived late at night, there appeared to be no need for operative attack until the following morning.

His head was large for his age. The anterior fontanelle was closed, but there was a marked cracked-pot sound on both sides. The pupils were large and barely reacted to light. The right disc was blurred. There was weakness of the right and left abducens muscles. He did not talk, but appeared to recognize his family. He had complete right-sided spastic hemiplegia and apparently anesthesia also. The deep reflexes were increased on the right side; there was a positive Babinski on the right. The abdominal reflexes were not obtained.

The ventriculograms which had been brought with the patient showed moderate grade of hydrocephalus on the right. The left ventricle did not fill. The third ventricle was oblique and together with the right lateral ventricle pushed markedly toward the right. It was evident that there was a left cerebral tumor (Fig. 14).

Gross necropsy report (Fig. 13). The greatest diameter of the tumor is 7.5 cm., the smallest 6 cm. It projects into a huge descending horn which is shut off from the remainder of the ventricular system. The surface of the tumor is quite

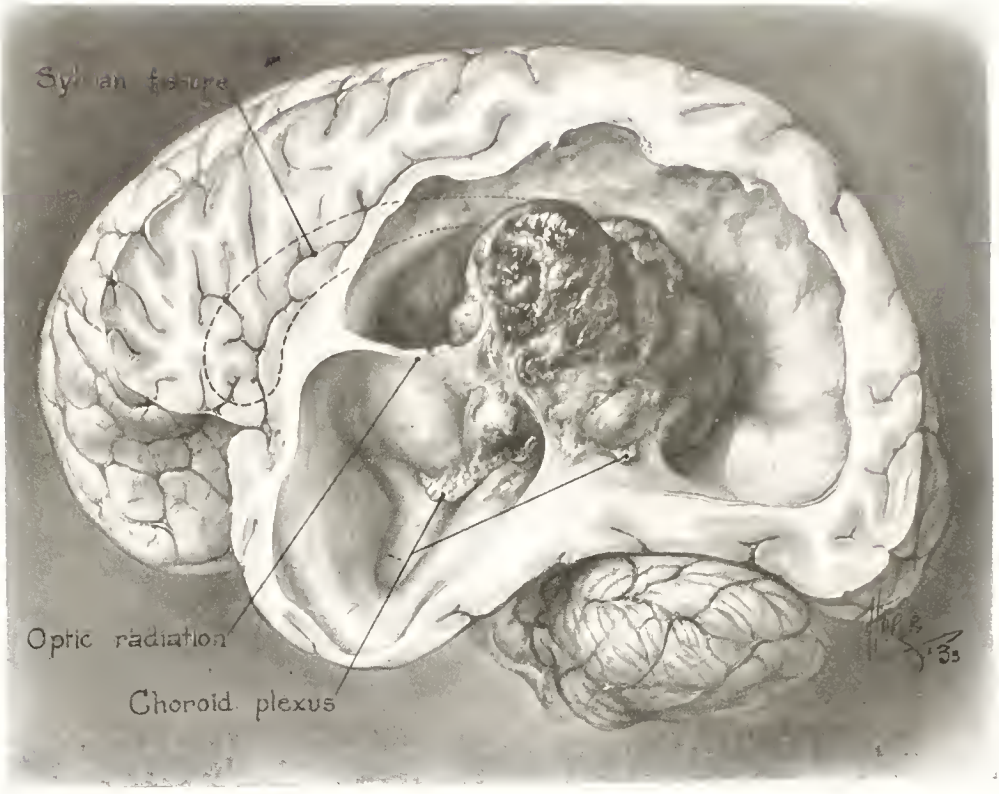

FIG. 13. Malignant adenoma of the choroid plexus. (Writer's Case I.)

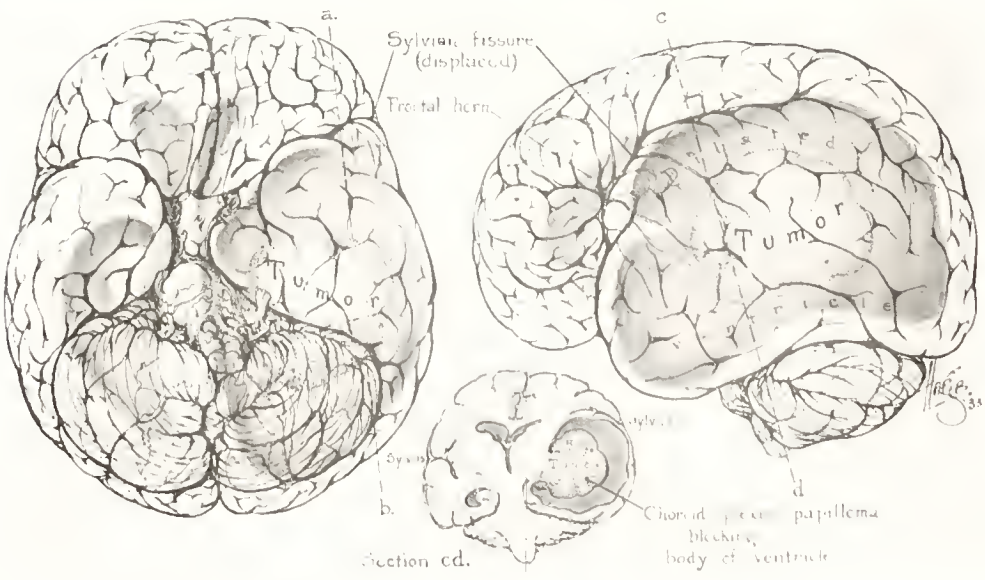

FIG. 14. Sketches showing the location of the tumor and its effect upon the ventricular system. The huge dilated ventricular cavity in which the tumor rests represents only the posterior and descending horns which are shut off from the remainder of the ventricular system by the tumor's obstruction.

nodular, eroded in part and covered by a yellow exudate and a small blood clot—doubtless the source of the fatal intraventricular hemorrhage. The tumor is highly vascular as evidenced by its deep brownish-red color. It is firmly attached to the floor of the descending horn over a very wide base and

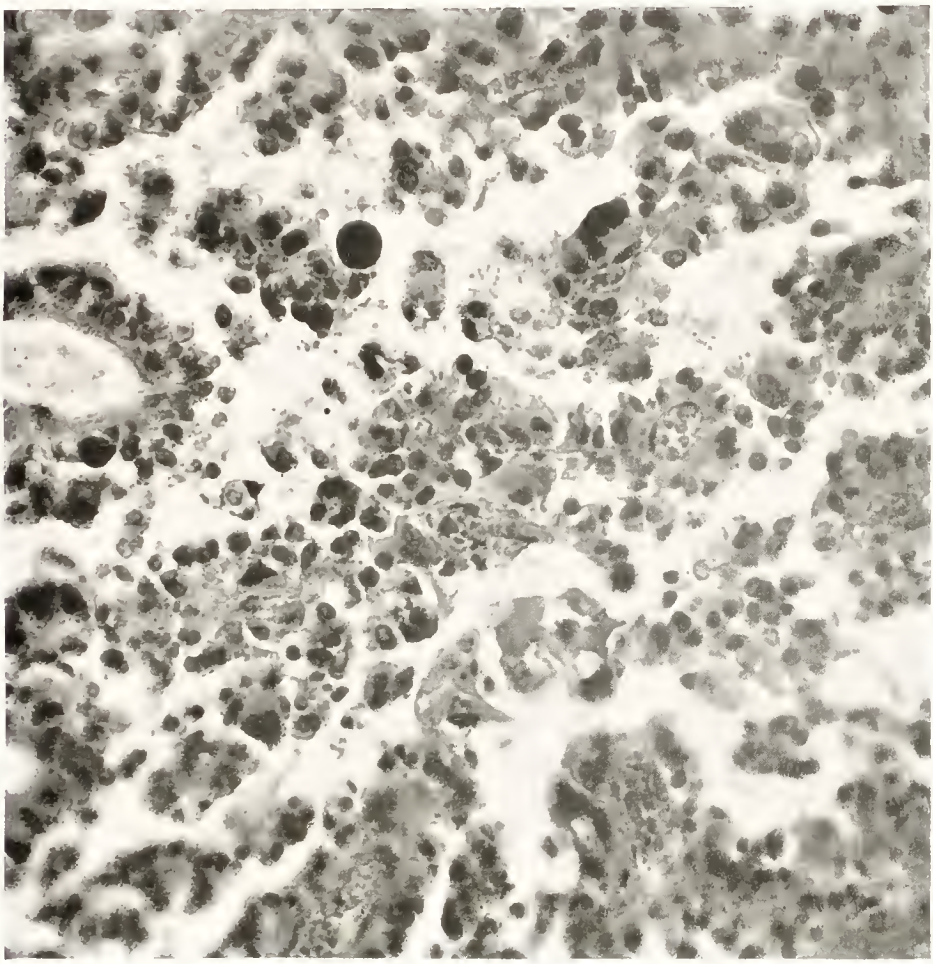

FIG. 15. Photomicrograph of malignant adenoma of the choroid plexus (Fig. 13)

though fairly circumscribed, is not encapsulated. Unfortunately the postmortem examination was restricted to the head. That metastases elsewhere were not present cannot be stated, but there were no symptoms to suggest them.

Microscopic note (*Fig. 15*). Although the papillary arrange-

ment of the choroid plexus is evident in patches throughout the sections, the cells are nowhere uniform but are of all shapes and sizes; there are many giant cells of varying size. In many places the cells are closely matted together without any tendency toward alveolar formation. There are many round goblets of varying size, some within large cells, others without and much larger than the largest giant cell. This material is doubtless a secretion. Many areas of necrosis are present throughout all the sections.

Had the papillary arrangement of the tumor been absent, it would not have been possible from our present knowledge of intracranial tumors to have made a diagnosis of a tumor of the choroid plexus from the remaining cells.

Case II

D. A. Admitted: January 27, 1933. Discharged: February 10, 1933. Readmitted: July 1, 1933. Discharged: July 30, 1933.

Patient aged $3\frac{1}{2}$ years referred from the Department of Pediatrics of the Johns Hopkins Hospital with the diagnosis of an unlocalizable brain tumor.

Complaints. Headache and vomiting of four weeks' duration.

Family history. It is worthy of note that two brothers died at the ages of two and five months. In each instance death was sudden and of unknown cause. One sister is living and well; another sister died of dysentery. Mother is 25 years of age, father 32; both are well.

The *past history* is negative. Birth and development were normal; talked at ten months and walked at sixteen months; scarlet fever at the age of two.

Present illness. Began four weeks ago with vomiting and headache. Attacks of vomiting and headache have since occurred almost daily. There have been no other symptoms, except perhaps on one occasion when he was thought to have staggered to the right.

Physical examination. The child responds normally but is quite drowsy. The head is definitely larger than normal, measuring 15.5 cm. in circumference. A marked cracked-pot sound is elicited on tapping the region of the fronto-parietal suture line on either side.

Neurological examination. There is a mild grade of papilloedema on both sides, a little greater on the right. The neck is definitely stiff. The deep reflexes are slightly hyperactive, but equal on the two sides. Other neurological signs are absent.

Lateral x-ray of the head shows generalized convolutional atrophy and separation of the fronto-parietal sutures on both sides.

Impression. Unlocalizable tumor, perhaps in the posterior cranial fossa.

This localization was suspected:—first on the law of probability; second from the x-ray changes which were suggestive but not pathognomonic of hydrocephalus; third, the cervical rigidity; and fourth, the possible history of having staggered on one occasion.

Ventriculography, January 27, 1933. Patient was anesthetized for ventricular estimation and for a cerebellar operation should this test show enlarged ventricles on both sides. However, neither ventricle could be reached; this definitely excluded a cerebellar tumor. Patient was then removed from the operating table and a spinal air injection performed. Only 25 cc. of fluid could be removed, and 20 cc. of air injected. The shadow of the fourth ventricle was normal, but the air did not pass beyond the aqueduct of Sylvius. The cisterna chiasmatis contained air but its size was markedly diminished. This injection was, therefore, without help in localizing the tumor. Excepting a slightly greater papilloedema in the right fundus, there was not the slightest clinical evidence by which the tumor could be localized to either cerebral hemisphere. Perforator openings were then made over the anterior horns of the ventricles and the left ventricle was punctured; 10 cc.

of fluid spurted under great pressure and an equal amount of air was injected. The ventriculograms showed the left ventricle to be markedly pushed to the left side; the third ventricle did not fill nor did the right lateral ventricle. It was evident, therefore, that the tumor was in the right cerebral hemisphere.

Operation January 28, 1933. Since the right ventricle did not contain air there was no indication of the exact site of the

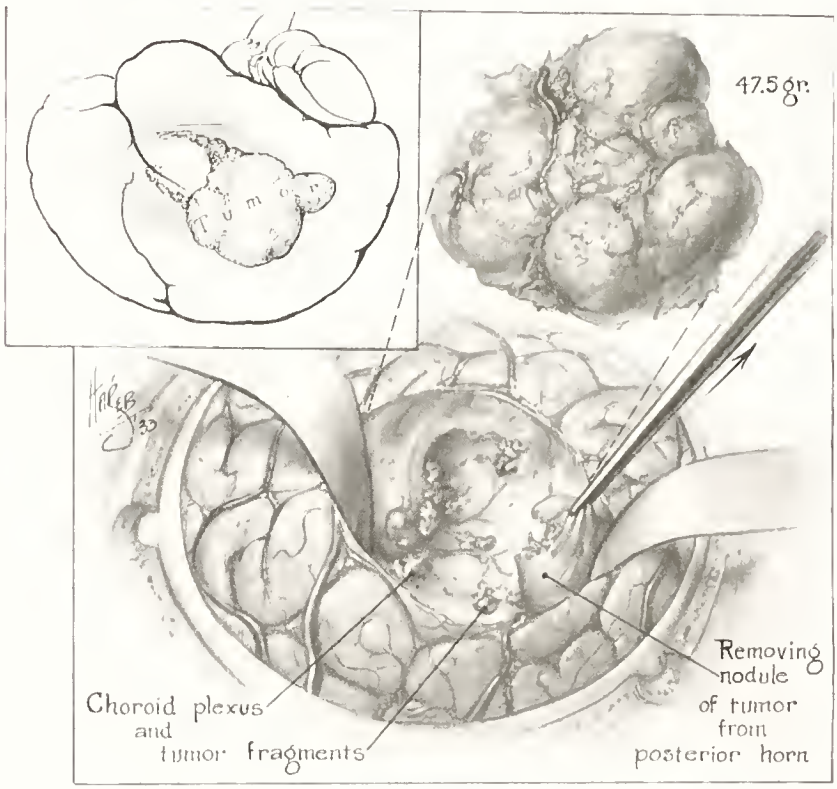

FIG. 16. Operative sketch of malignant adenoma in the choroid plexus (Case II). This tumor recurred six months later.

tumor in the right hemisphere. It was, therefore, necessary to turn down a large bone flap. The dura was under great pressure. About 3 cm. posterior to the Rolandic vein there was definite indication of an underlying tumor for the convolutions were wider, flatter, and paler than elsewhere. A ventricular needle encountered resistance at a depth of 4 cm. A transcortical incision was then made and the surface of the tumor

was seen at the indicated depth; it was reddish-brown and seemingly encapsulated. A circular area of silent cortex (15 grams) was excised over the posterior part of the growth. The tumor was quickly extirpated with the finger (Fig. 16). When bleeding was controlled, the bed from which the tumor had been removed was seen to be the walls of the lateral ventricle. The choroid plexus and the body of the ventricle were in full view. It was then seen that part of the tumor had broken off and the remaining portion filled the posterior horn of the ventricle. This was quickly extirpated with the finger. The descending horn was now in view and the roughened walls indicated that the tumor had extended into the posterior half of this part of the ventricle. An additional small area of the tumor was seen attached to but easily removed from the inferior wall of the descending horn near its junction with the posterior horn. The glomus of the choroid plexus could not be seen. It had apparently been removed with the tumor. Elsewhere the plexus of the body and descending horns was normal.

As soon as the tumor had been extirpated its character made us feel that it probably was a tumor of the choroid plexus. It was well encapsulated and though very cellular, was quite solid and firm. Its deep brownish-red color and its cellular character resembled very much the picture of a cellular dural endothelioma, which, of course, it could not be because of its position. From its gross character and its position in the ventricle, a gross diagnosis of a choroid plexus tumor was made. The weight of the tumor was 47.5 grams. There was ample room to permit closure of the dura and replacement of the bone flap.

The patient made an uneventful recovery and left the hospital two weeks following the operation. There was no motor or sensory loss at the time of discharge.

Six months later patient returned with signs of intracranial pressure, but still there were no localizing signs. Not knowing whether the tumor had recurred locally or a metastatic nodule

was located elsewhere, ventriculography was again performed. A huge right ventricle was encountered, and 150 cc. of air were required to replace the extracted fluid. Such a large ventricle suggested a cerebellar tumor, but the ventriculograms showed a tremendous cavity (greatly dilated descending horn) which did not communicate with the remainder of the ventricular system.

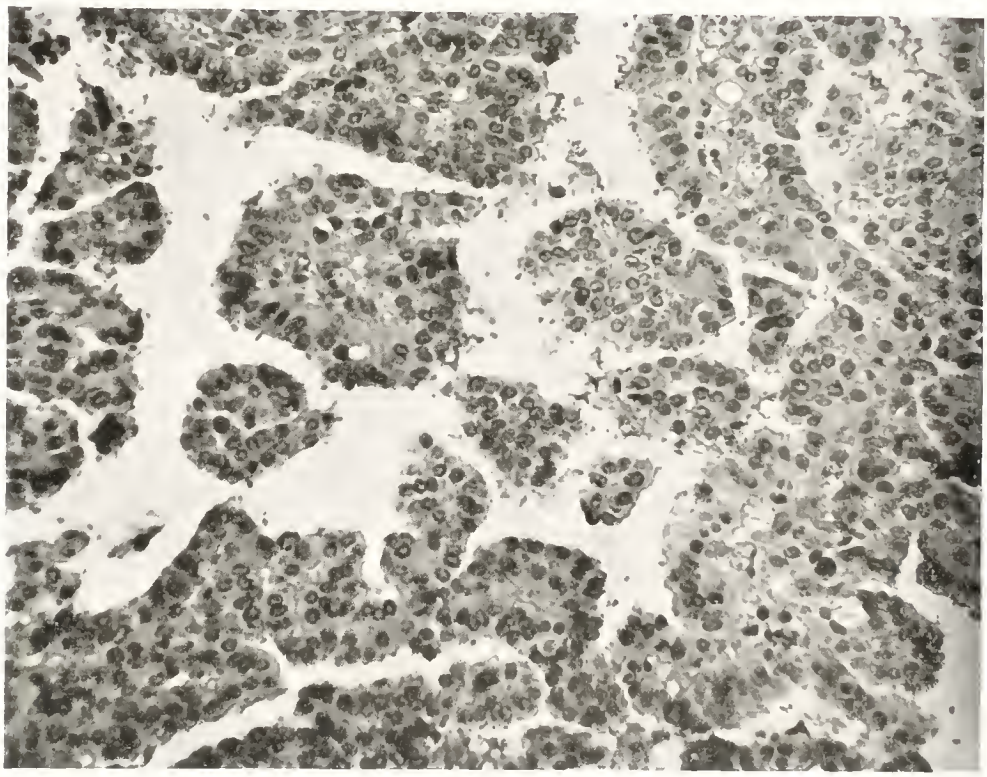

Fig. 17. Photomicrograph of adenoma (Fig. 16) showing the microscopic appearance of the choroid plexus. There is much more regularity in the size and arrangement of the cells than in the preceding adenoma (Fig. 15).

Operation revealed a large mass in the occipital lobe; it blocked the body of the lateral ventricle and caused hydrocephalus which was restricted to the descending horn. Cure of the tumor was regarded as impossible, though it was partially removed. Upon further reflection it was thought to be advisable to give him a final chance of cure, though it must be

very slight, by resecting the entire occipital and posterior part of the parietal lobes. This was done July 12, 1933. The patient left the hospital August 1, 1933, just as this note is being written.

With respect to the possibility of secondary tumors developing in the ventricular walls, it is worthy of note that none were present in the wide expanse of the huge descending horn.

Microscopic note. The histological appearance of the tumor (Fig. 17) is much like that of the preceding case (I). There are, however, fewer of the wild cellular irregularities, including giant cells, and less of the secretory globules, although all of these digressions are plentiful.

Dr. Arnold Rich was consulted about the histological appearance of the above two tumors, together with a third tumor of the plexus known to be benign. He at once concluded that the cellular irregularities and the giant formations were evidence of the malignancy in the above two tumors. These changes were absent in the benign tumor. Although the secretory globules were present in the benign tumor also, they were far less numerous than in the malignant growth. Whether this is significant or not he was unable to say.

CHAPTER IV

THE LARGER ENCAPSULATED TUMORS CAUSING SYMPTOMS

In all but one of the twenty-five benign encapsulated tumors assembled from the literature the neoplasms have been found at necropsy. From the varied nomenclature employed at different periods it is not always possible to determine the exact character of the tumor—a difficulty still further increased because, in most instances, the microscopic descriptions are meager. Several of the earlier tumors are described as sarcomata, but as this is an exceedingly rare lesion in the brain it is assumed in view of their actual or seeming encapsulation to be either ependymal gliomata or ependymomata. An effort to group these tumors, therefore, would be open to such serious doubts that it seems better to comment upon their probable or possible character when our own cases are presented. However, several unusual tumors are present among the cases from the literature. For example, in Henning and Wagner's case (1856) the tumor was made up of "masses of cartilage and bone surrounded by fluid and blood clots all contained within a closed membrane." Since it did not contain the other elements of a dermoid, epidermoid or teratoma, the authors were forced to call it a "fetal enchondroma."

Cayley and Brown's cyst, also occurring in an infant, is remarkable in that the cyst sprang from the glomus of the choroid plexus and pushed between the cerebral hemispheres reaching the dura. Its great size caused asymmetrical enlargement of the head. The description of the very thin transparent walls and clear serous fluid content would lead one to regard it as a cyst of the choroid plexus, and yet if this is true, it is the only example of a symptom producing cyst that I have been able to find.

Broca's tumor, weighing 25 grams, is apparently made up of bone with definite canaliculi (microscopic report). It dangles freely in the ventricle being attached to the wall by a pedicle and is covered by a smooth membrane.

WRITER'S SERIES OF TUMORS CAUSING SYMPTOMS

From the writer's surgical experience during the past fifteen years (since the advent of ventriculography) fifteen (apparently) benign encapsulated, enucleable tumors have accumulated. All have been disclosed at operation, and fourteen have been totally removed; the remaining case was exposed but not enucleated.

Although not a common tumor, it nevertheless is one which has been encountered on an average of once a year (there have been three in the current year, and two of these in a single month) and in perhaps 1 per cent of all intracranial tumors. That the number of cases in this series is relatively far greater than the number from the literature (in a given period of time) is due to the intensive study of brain tumors by ventriculography.

Case I

W. B. Age 23. Admitted: August 6, 1918; September 9, 1918. Total enucleation of tumor. Discharged: August 16, 1918; December 8, 1918.

Patient was referred by Dr. Joseph D. Buxton, Newport News, Virginia, August 6, 1918, with the diagnosis of brain tumor.

Family history and past history negative.

Complaints. Headaches, vomiting spells, deafness in the right ear, dimness of vision, more pronounced in the left eye.

Present illness. Patient dates his present illness from April, 1917, sixteen months before his admission to the Johns Hopkins Hospital. His first symptom was diplopia; in two weeks this disappeared and has never returned. However, on questioning, patient recalls that he has had periodic head-

aches for the past three or four years; associated with these headaches were spells of vomiting. Unquestionably these headaches are part of his present illness, and the duration of his present illness dates back at least four years. These periodic headaches were located in the forehead, on top of the head, in the occipital region and back of the eyes. About three weeks before the onset of diplopia he had paralysis of the left side of the face. He says he was unable to close his left eyelid (since there was no facial paralysis he was questioned carefully to determine whether the facial paralysis might not have been actually on the right side and due to the involvement of the left Rolandic area). There was also numbness on the left side of the face extending exactly to the midline. Tactile tests made at that time showed a reduction in the perception of sensation on that side. After two weeks both the sensory and motor changes gradually improved, although a definite hypoesthesia is still present on the left side. About the time of the facial numbness and weakness headaches became much more severe and vomiting more frequent. He has vomited as much as eight times in a single day. On one occasion he vomited while fast asleep; his mother tells him that the vomitus was thrown across the room. The headaches would last two or three days and then disappear. He is quite certain that at this time his hearing was not affected in either ear. He was in the telephone business and it was necessary to use both ears. The vision in the left eye began to decline about the time of the facial paralysis and numbness.

Fourteen months ago, that is about two months after his spell of diplopia, a cerebellar operation was performed elsewhere. He knows nothing of the operative findings. He insists that the deafness in the right ear followed this operation and that it was not present before that time. No improvement has followed during the period of more than a year since the operation. He has steadily lost strength, his memory is not so good; there is some loss of smell and taste. He has had no motor or sensory disturbances other than the attack mentioned above.

Eight months ago he became unconscious and remained so for four days. He knows of no convulsion that occurred at that time. After returning to consciousness his condition was about the same as before. His headaches have not been so severe since his operative procedure, but they have been closer and closer together during the past two months, and during this time the patient has gradually become weaker. There have been aching pains through both hips and legs; pains are steady and seem to alternate from one side of the body to the other; at the present time they are worse on the left side.

Following the operative procedure a large swelling was present at the back of the neck at the site of the operation (a bilateral cerebellar operation had been performed). He insists that this swelling after being present for several months entirely disappeared and returned only two months ago, and that since its return it has not been as full and tight as formerly. He also says that its size varies from time to time.

Physical and neurological examinations. A fairly well nourished young man of 23, rather listless and depressed. Shows evidence of loss of weight of about 20 pounds. Visual fields show a marked reduction both for form and color in the left eye; there is a suggestion of a nasal hemianopsia for form and probably definitely for color, although with the marked contraction of the visual fields one could scarcely look upon this as being definite; the field of vision in the right eye is entirely normal. Visual acuity 10/40 left; 10/20 right. There is papilloedema of about three diopters in each eye ground; the veins are full and tortuous. There is fine rapid nystagmus on looking to the right and coarse nystagmus on looking to the left; slight weakness of the right external rectus muscle. There is definite, but slight, hypoesthesia for touch, pain, heat and cold on the left side of the face. Corneal reflexes are equal. There is no facial asymmetry; no motor disturbances on either side. The right ear is totally deaf; on the left side the hearing is within normal limits and air conduction is greater than bone conduction. There is a probability of nucinate attacks because he complains of smelling and tasting things

the nature of which he cannot describe. There is slight ataxia on finger-to-nose test; slight tendency to sway to the right when standing with the feet together and eyes shut. The gait is staggering. There is no adiadochocinesia. The reflexes are normal. X-rays of the head are negative.

With these findings, I still thought the lesion was probably cerebellar. It was difficult to believe that a total deafness on the right side could have been the result of the previous operation, of which I had no information. The numbness of the left side of the face was quite definitely of peripheral origin. We thought his tumor was in the cerebellum.

The patient decided not to be operated upon and left the hospital, returning, however, a month later in essentially the same condition, except that headaches had been practically constant and vomiting had been more frequent. Re-examination disclosed no new findings. The suboccipital swelling was full and tight.

Operation, September 10, 1918. The old cerebellar incision was reopened and the cerebellar region thoroughly exposed, but no evidence of a tumor could be found. The post-operative course was uneventful.

Ventriculography. At this time we were experimenting with ventriculography. The patient was advised that there was a possibility that something might result to his advantage if he were willing to have the test made, but nothing could be promised. He was anxious for any effort and accordingly on October 23, 1918 ventriculography was performed for the first time on a tumor suspect. The right ventricle was tapped and 35 cc. of fluid removed and an equal amount of air injected. The ventricles were perhaps double the normal size; the right ventricle was of normal shape; the left ventricle was equally normal until the air reached the posterior part of the body of the ventricle where it stopped abruptly along a sharp curved line (Fig. 18). The posterior and descending horns did not fill with air; the tumor, therefore, was localized in the posterior horn of the left lateral ventricle.

Second operation, November 5, 1918. A bone flap was turned down in the left occipital region. A small portion of a reddish-brown tumor protruded through the cortex well posteriorly and all of the contiguous brain was very pale and soft, indicating an extensive subcortical extension of the tumor. The exposed part of the tumor was then followed into the sub-

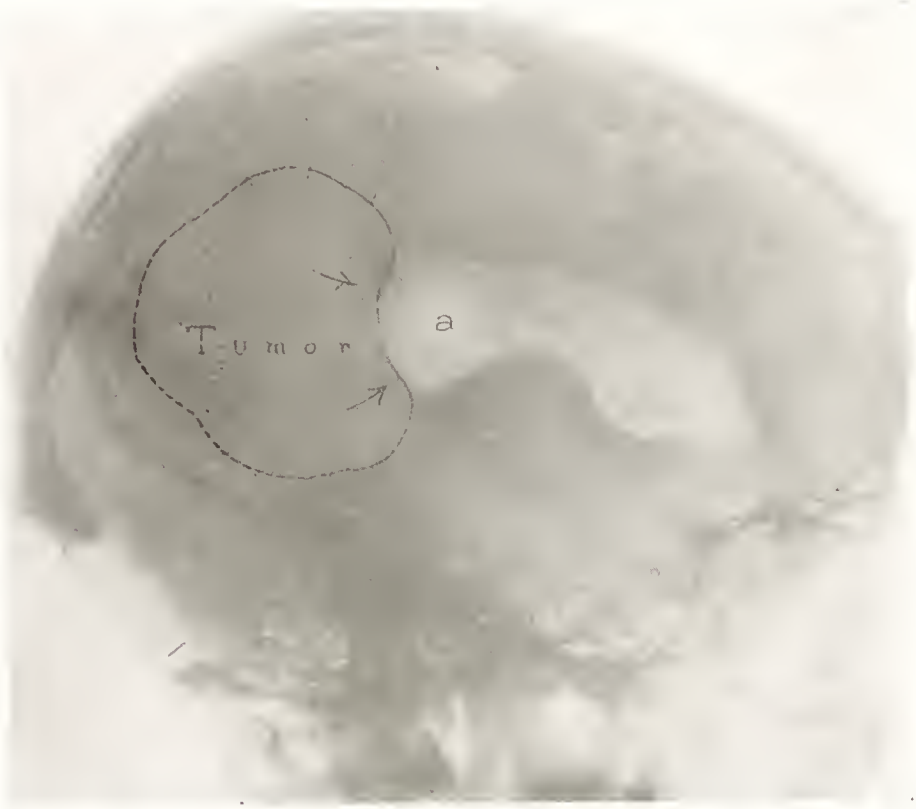

FIG. 18. Ventriculogram from which the localization was made. The left lateral ventricle terminates abruptly at the sharp curve line which represents the anterior border of the tumor. The dotted line indicates the position of the tumor.

cortex and everywhere it was found to be well encapsulated. The thinned superimposed brain tissue easily separated from the margins of the tumor and as the separation of the growth continued into the depths the wall of the lateral ventricle came into view and was opened. The tumor was found to be adherent to the glomus of the choroid plexus, but was easily

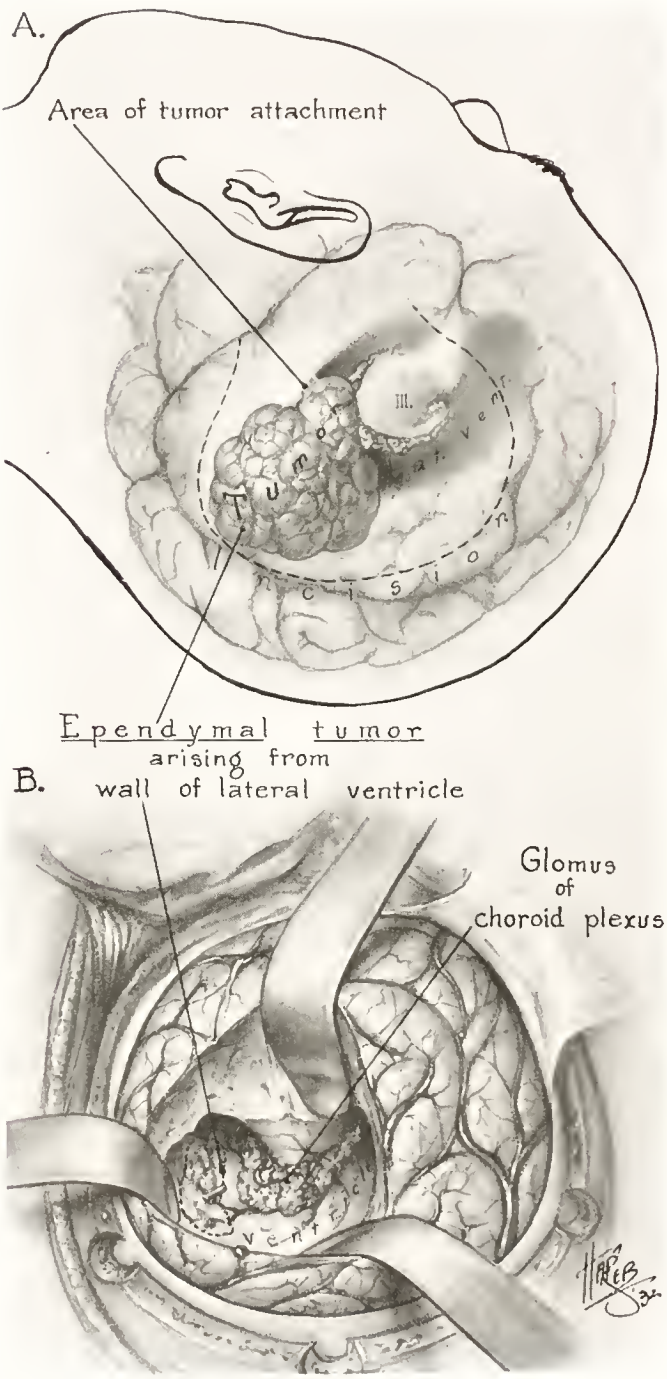

FIG. 19. Operative approach of tumor in Case I

separated from it. The tumor was firmly fixed to, fused with, and arose from the ependymal lining of the ventricle along the posterior wall of the descending horn and the inferior wall of the posterior horn. This portion of the wall of the ventricle was excised as a precaution against recurrence. The tumor

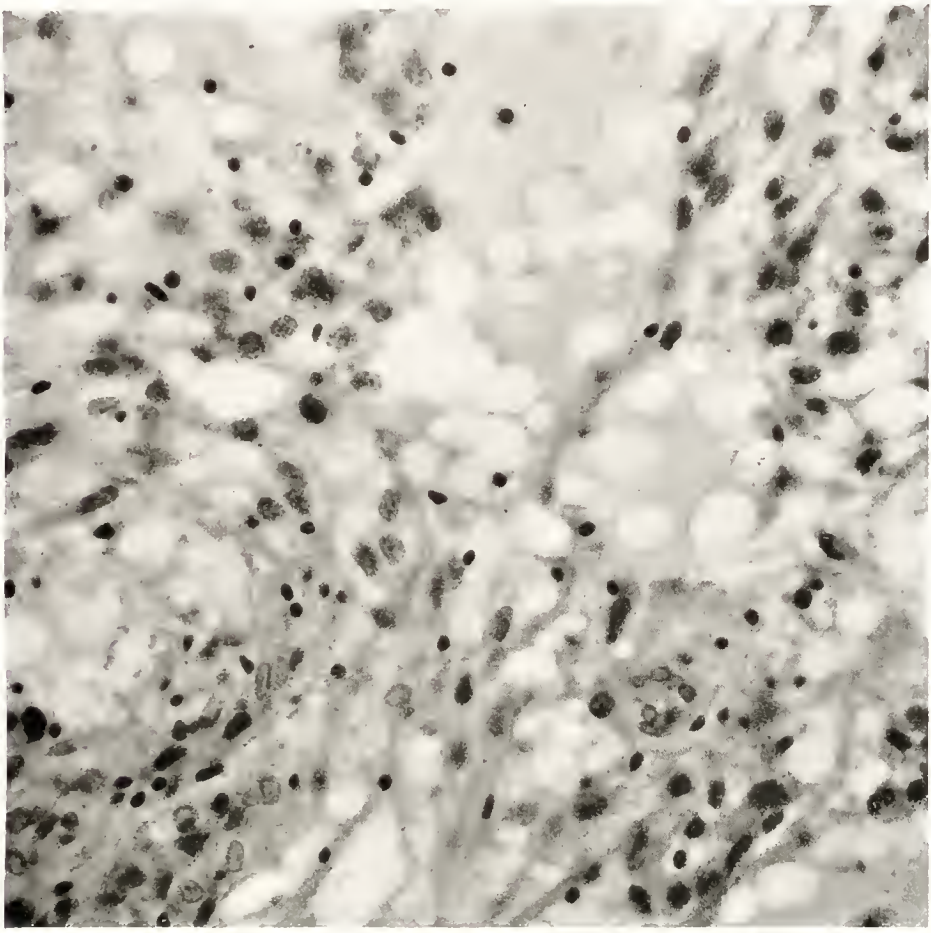

FIG. 20. Photomicrograph of tumor, Case I

filled much of the descending horn, the inferior horn, and the beginning of the body of the ventricle (Fig. 19).

There was very little bleeding during the entire removal of the tumor; this was controlled by a few silver clips and by application of cotton immersed in warm Ringer's solution. When hematosis had been effected the dura was closed, the

bone flap replaced and the galea and skin closed with interrupted sutures of silk.

The tumor was encapsulated, soft and nodular, and definitely had its origin from the ependymal wall of the lateral ventricle. The interior of the tumor contained a round smooth-walled cyst (1.5 x 1.5 cm.) filled with yellowish fluid.

FIG. 21. Glial stained showing patch of glial in the tumor, Case I

Post-operative course. The post-operative course was uneventful, the patient leaving the hospital December 8, 1918.

Subsequent course. Patient is still living and is well (15 years after removal of the tumor). For a number of years he had occasional convulsions, but these have been absent for the past five years.

Comment. This case was a very fortunate beginning in the field of localization of intracranial tumors by ventriculography, which will play such an essential rôle in the detection of subsequent tumors of this group.

Microscopic report. Connective tissue with many nuclei and rather heavy fibrous strands. In places the fibrous tissue is compact. In other places there is a loose areolar arrangement and the spaces are filled with globules of pink staining colloid (Fig. 20), but nowhere are there any cells of epithelial character. In one place the tumor grows out from the brain tissue with no suggestion of a dividing line. In this region there are many calcified areas. There is no epithelial lining on the surface of the tumor, only a thin wall of connective tissue. There are occasional patches and one large strand of glial tissue (Fig. 21).

Probable diagnosis. Ependymal fibroma or glioma.

Case II

C. M. Age 23. Admitted: August 23, 1920. Total enucleation of tumor. Discharged: December 15, 1920.

Referred by Dr. R. B. Day, Pendleton, South Carolina, August 23, 1920.

Complaints. Headache and failing vision.

Family history and past history. Negative.

Present illness. Began four months ago with severe headache in the right frontal region, sometimes extending to the left frontal region. Headaches have occurred almost daily since the time of onset; they are always present when he awakens in the morning and they wear themselves out in about three or four hours. At times his head feels as though it "were going to burst wide open"; again it is of a throbbing character and at other times a burning sensation. When he stoops the headaches are brought on for a short time; this has been so marked that he has had to give up work which caused him to bend over. Associated with stooping also is nausea, but he has had no vomiting. The headaches have not been severe enough to keep him in bed. One month ago while driving a wagon a

"sudden glare came into his eyes." It seemed as if there were something in front of his eyes and he tried to wipe it away, but without success. This has been growing steadily worse so that he is now able to read but little. Both eyes are equally affected. A few days after the visual disturbance he noticed that when he was recumbent a "buzzing" developed in his right ear, not in his left. It ceased as soon as he assumed an upright position. The buzzing was as if some insect were trying to get out of his ear. Only for the past two days has this buzzing been absent. Diplopia developed a month ago, but this persisted only for a few days and has never returned. About a week ago there was staggering for a short time, but this has never returned. There has been no dizziness and no convulsions. He complains of slight weakness of the left side, but there is no objective loss of motor power. There have been no sensory disturbances, except perhaps that he has had a tingling sensation across the hair on the right side, but not on the left.

Physical and neurological examinations. Patient is a well developed young man, appearing normal both mentally and physically. There is a moderate grade of papilloedema in both discs. The visual acuity is reduced to 2/100 in the right eye and 50/100 in the left eye. Visual fields are normal. The neurological examination is otherwise entirely negative. There is no disturbance of the extra-ocular muscles; no nystagmus; no motor or sensory loss. His gait is normal; Romberg negative and reflexes unchanged.

X-ray of the head is negative.

Clinical impression. Tumor in the right cerebral hemisphere, perhaps in the frontal lobe.

Ventriculography, August 27, 1920. The left ventricle was tapped, but only a small amount of fluid was obtained. It was under great pressure. About 15 cc. of air was injected. The ventricular system was dislocated towards the left side; both ventricles were very small. The left ventricle seemed perfectly normal (Fig. 22, *A*). Air in the right ventricle extended

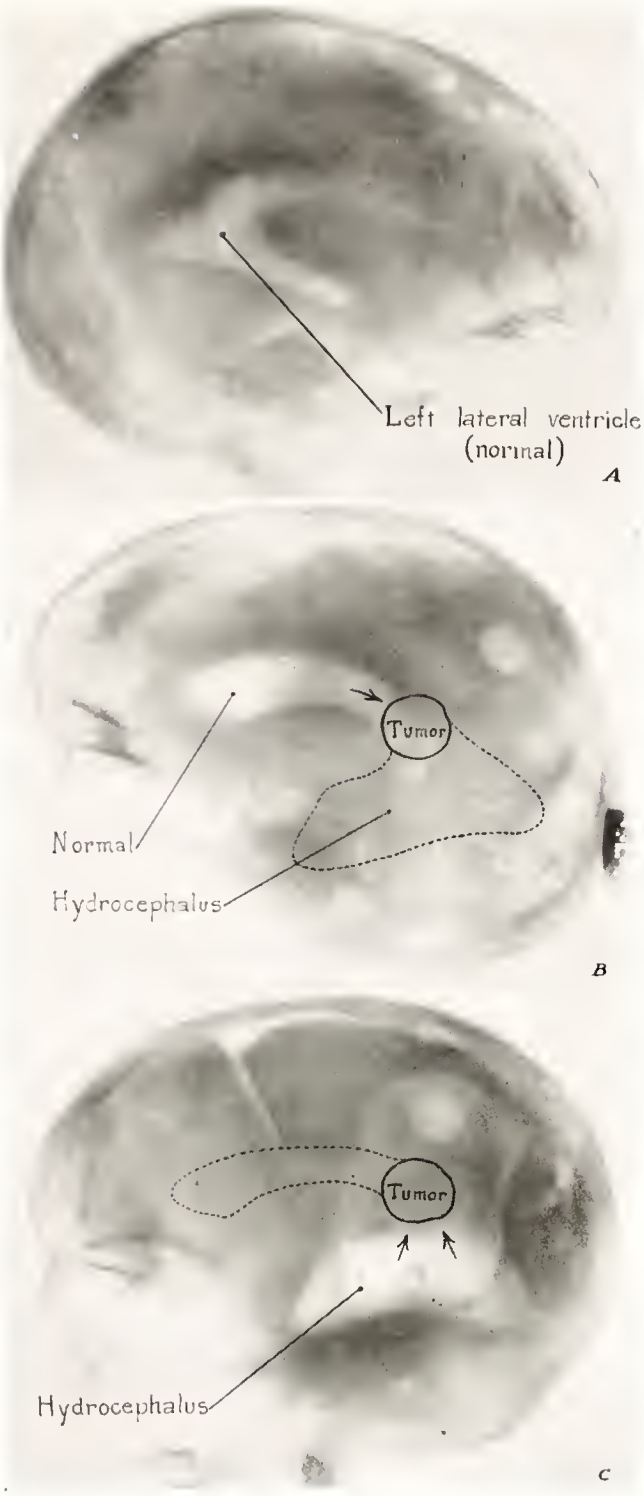

FIG. 22. Ventriculograms of Case II.

A. Normal left ventricle.

B. Air terminates in the posterior body of the right lateral ventricle, that is, at the anterior border of the tumor.

C. Air injected into the dilated descending horn of the right lateral ventricle; it terminates at the posterior border of the tumor; the tumor, therefore, lies between the triangular shadows in *B* and *C*.

to the posterior part of the body of the ventricle, but did not fill the posterior or descending horns (Fig. 22, *B*).

Operation, September 1, 1920. Under ether anesthesia a large bone flap was turned down on the right side exposing the parietal and occipital regions. Aside from the fact that the temporal and occipital lobes may possibly have been slightly softer and paler than elsewhere, no abnormality was seen. Thorough exploration of the brain failed to reveal any evidence of a tumor and the wound was closed. At the same time a large decompression was made in the temporal region.

The patient was kept in the hospital for several weeks because we were reluctant to let him go with such a tight bulging decompression, and we were hoping that we might yet be able to find the tumor. His headaches continued despite the large decompression.

Ventriculography, October 26, 1920. Ventriculography was again performed. On this occasion the right ventricle was tapped and easily reached; it was much larger than the left ventricle had been at the time of the previous injection. Twenty-five cubic centimeters of fluid were easily withdrawn and an equal amount of air injected. This air remained confined to the posterior and descending horns of the ventricle, both of which were much enlarged. The air stopped abruptly at the posterior part of the body of the ventricle (Fig. 22, *C*). In checking with this former plates it was evident that the anterior border of this air-shadow was about 2 cm. behind the posterior limit of the air-shadow from the earlier injection. A tumor must, therefore, be located in the lateral ventricle between these two extremes of the air-shadows. Moreover, the tumor was causing local hydrocephalus up to the point of obstruction and it was the dilated part of the ventricle which was causing the intracranial pressure and the bulging decompression.

On October 27, 1920, patient had a convulsion beginning on and chiefly involving the left side of the body. After a time the right side was also affected. The eyes and head were turned

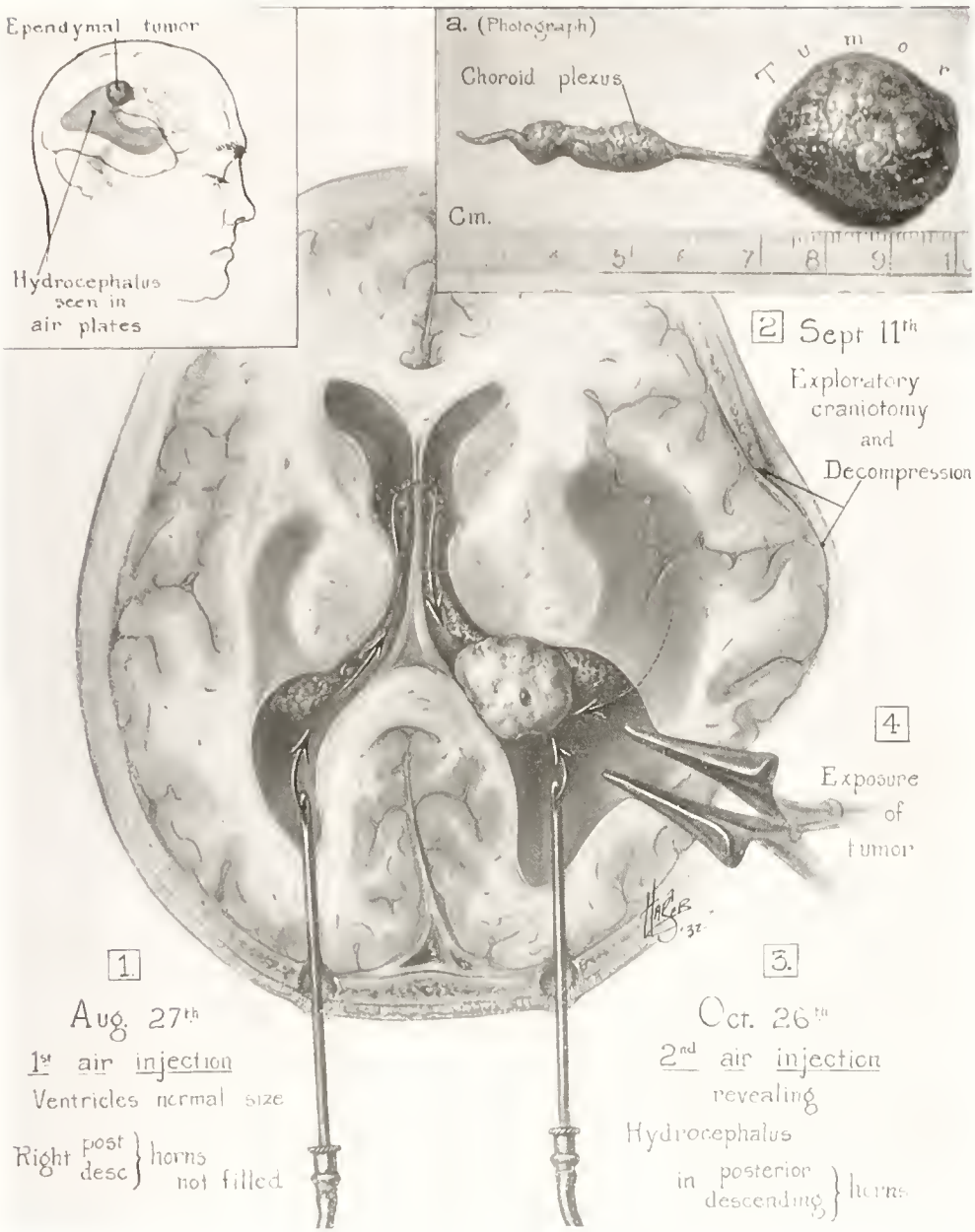

FIG. 23. Drawing showing position of tumor as disclosed at operation and its effects upon the ventricular system, from which the diagnosis was made by ventriculography. The upper right inset is a drawing of the tumor removed at operation and the choroid plexus attached.

to the left. One week later he had two similar convulsions. It is very probable that these convulsions were induced by the

air injection. Since the attacks there has been marked weakness of the left arm and leg. There was no anesthesia.

Operation, November 15, 1920. From the ventriculographic findings there could be no doubt of the exact position of the tumor in the posterior part of the body of the right lateral ventricle. The old wound was reopened; a nasal dilator was passed through the cortex into the posterior horn of the lateral ventricle. When the ventricle was reached the tumor could not be seen, but as the blades of the nasal dilator were directed farther anteriorly the posterior border of a well encapsulated reddish-colored tumor came into view (Fig. 23). The tumor lay at a depth of 6.5 cm. below the surface of the brain, and at this great depth it was impossible to extirpate the tumor with instruments owing to an inadequate exposure. The nasal dilator was withdrawn and the finger inserted into the opening which had been produced. It was realized that the tumor lay alongside the internal capsule, but the finger easily surrounded the tumor with little trauma and it was enucleated from its bed with surprising ease; the choroid plexus was attached. There was some bleeding which was soon controlled by packing with moist cotton. When the ventricle was dry the wound was closed. The dura was sutured, the bone flap replaced and the cutaneous incision closed without drainage.

The patient left the hospital December 15, 1920, one month after removal of the tumor. Left-sided hemiplegia followed the operation. There has been only partial recovery of this function, patient walking with a decided limp. There have also been occasional convulsions. At the time of this note, September, 1933, the patient is still living and well.

When the tumor was inspected it was found to be almost spherical and weighed 9 grams (Fig. 23, inset). It measured 4.5 x 3.5 x 2 cm. Sections of the tumor show a fairly uniform firm, fibrous texture. There is a small cyst at the center.

Microscopic note. The general histological appearance is very much like that of Case I. The tumor is made up of fairly

loose connective tissue (Fig. 24). There are numerous cellular nests, which appear to be more compact areas of connective tissue. Dr. Rich, who kindly examined the sections, thought these cell clusters represented embryonal cells. The tumor is surrounded by a thin layer of compact fibrous tissue; there is

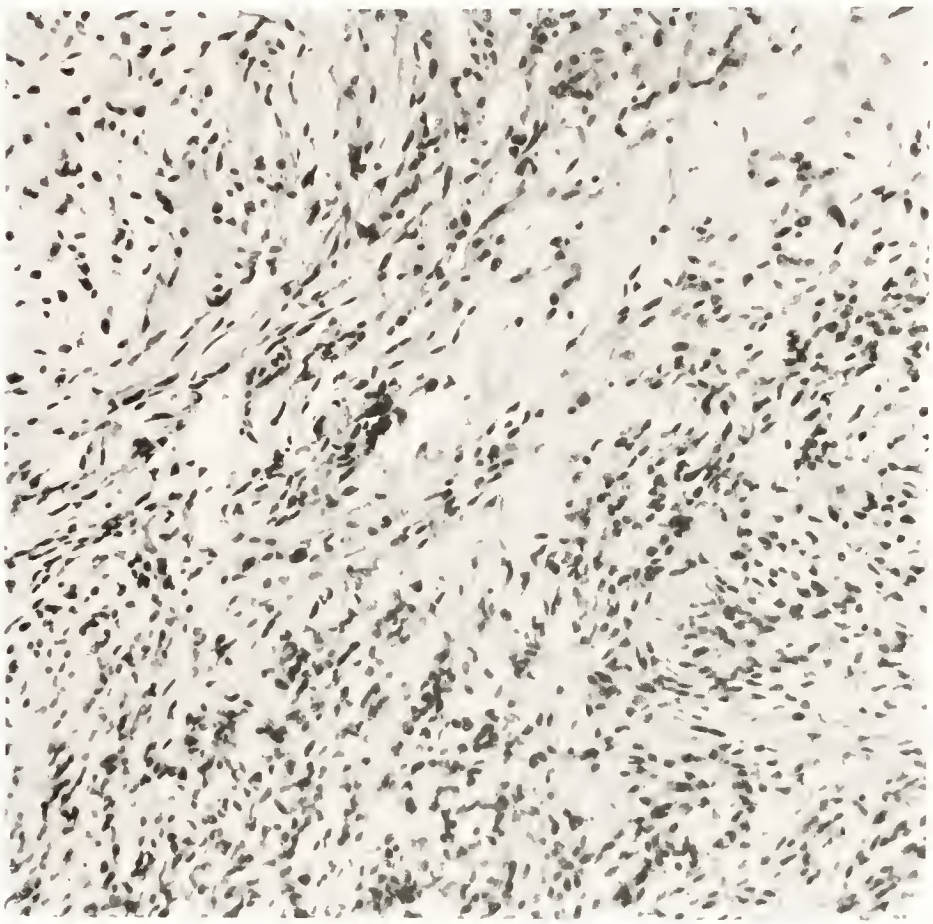

Fig. 24. Photomicrograph of tumor Case II

no epithelial lining. A small area of compressed choroid plexus is attached to the outside of the capsule; nowhere does it enter the tumor. A few strands of glial fibers are just beneath the capsule.

Diagnosis. Ependymal fibroma.

Case III

H. H. F. Age 12. Admitted: December 7, 1920. Cerebellar exploration, December 11, 1920. Death same day.

Referred by Dr. Harrison of Middle River, Maryland, with the diagnosis of a brain tumor.

Complaints. Headache and loss of vision.

Family history and past history. Negative.

Present illness. Began two years ago at the age of 10 when spells of headache, dizziness and vomiting developed. These attacks occurred about once a month, and in the interim he was perfectly well. Vomiting usually developed about an hour after the headaches and always relieved them. The headaches were always severe and always localized. It seemed "as though the top of his head was going to fall off." Between the monthly major attacks of headache there were minor spells lasting for only fifteen or twenty minutes and were not followed by vomiting. A year ago diplopia began; the eyes were crossed. About three months ago he had to leave school because of measles, mumps and influenza, all of which occurred in close sequence. Upon recovery from the influenza (about a month ago) the patient noticed that he had lost the sense of smell, and at about the same time his vision became quite poor; he could see only light and objects. He had had buzzing in his ears as long as he could remember. During the past three weeks the headache became constant and vomiting frequent. There have been no paralyses and no convulsions. There has been a nervous tremor in both hands for sometime. This has been noticed by his school teacher. There has been some difficulty in walking; he says he staggers, though not definitely to one side.

Physical and neurological examinations. Patient is a pale, undernourished boy. His mentality is apparently normal. The head is somewhat enlarged, but normal in shape and contour. There is a cracked-pot sound (Macewen's sign) which appears to be more marked on the right than the left side. He has only slight perception of light. The optic

discs are readily seen but are blurred, particularly on the nasal sides. The veins are full and tortuous. Smell is apparently entirely lost. There is perhaps slight weakness of the left external rectus muscle, but this is questionable. A definite, fine tremor of both hands is seen. There is adiadokocinesia of

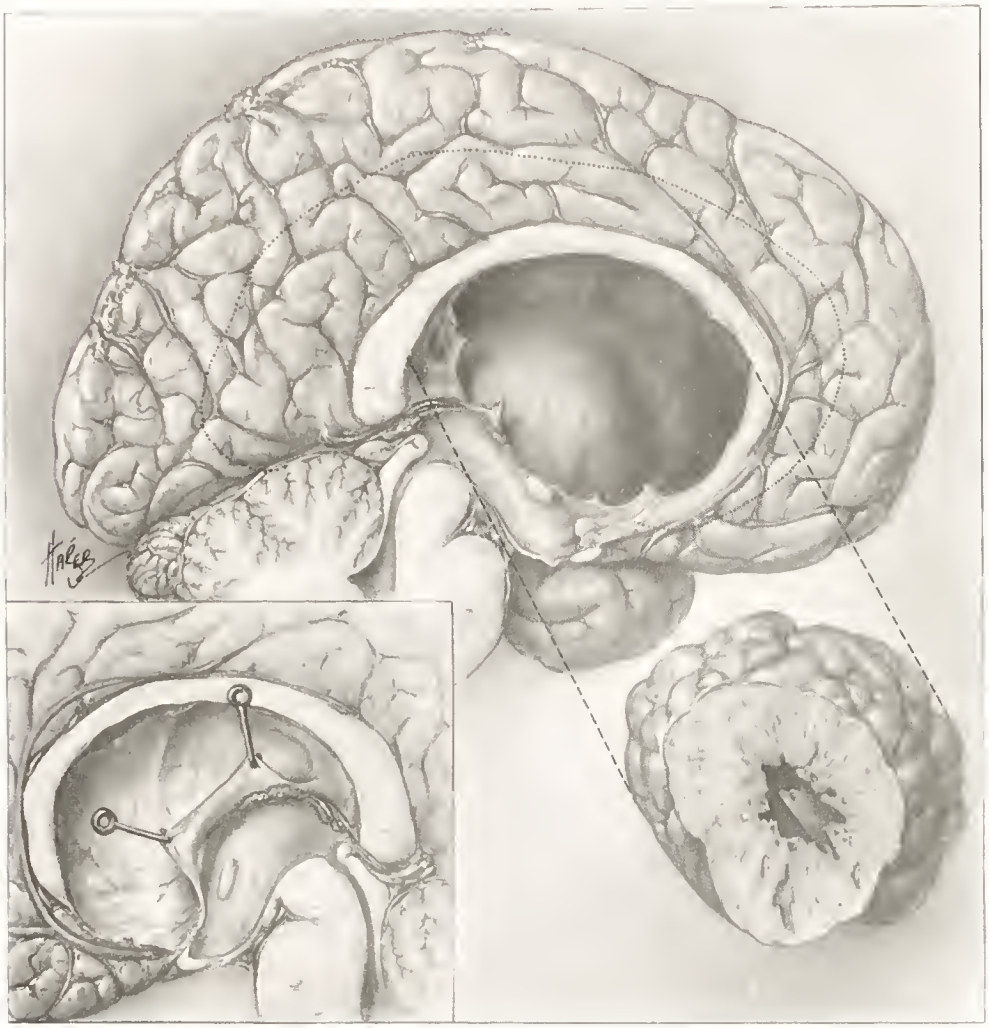

FIG. 25. Tumor from Case III

both hands, a suggestive though not definite ataxia, unsteady gait and a positive Romberg, with falling toward the right. The right abdominal reflex is absent, the left is normal. Knee-kicks and ankle-jerks are much exaggerated on the right side, but there is no clonus and no Babinski.

X-ray of the head shows advanced convolutional atrophy and separation of the cranial sutures.

Neurological diagnosis. Patient was shown in clinic, at which time he fell to the right side constantly when tested for Romberg. Because of the enlarged head, advanced convolutional atrophy, the history of staggering gait, adiadokocinesia and positive Romberg, and also upon the law of probability, the diagnosis of a cerebellar tumor was made. Even an air injection was not considered necessary, though it would have prevented a very bad mistake in diagnosis.

Operation, December 11, 1920. Under ether anesthesia a cerebellar exploration was made. The cerebellum looked normal; the cisterna magna was quite large. There was no herniation of the cerebellar tonsils into the spinal canal. The fourth ventricle was explored to the aqueduct which was found to be closed; a tumor was seen bulging into it. A small piece was removed for diagnosis. From the frozen section a diagnosis of glioma was returned from the laboratory. The wound was closed. Death occurred six hours later. Necropsy.

Gross pathology. The tumor was a very hard, nodular growth as large as a good sized dural endothelioma (Fig. 25) and was one of the largest tumors of the series. It occupied the anterior half of the left lateral ventricle and pushed over to the right side, obliterating the third ventricle and the aqueduct of Sylvius. A small cyst occupied the center of the tumor. The tumor was but slightly attached to the inner wall of the ventricle. Both lateral ventricles were greatly enlarged.

Microscopic sections of the tumor cannot be found, and the gross specimen, which was drawn immediately after necropsy, has been lost.

Diagnosis. Possibly an ependymal fibroma.

Case IV

S. J. Age 30. Admitted: October 5, 1925. Craniotomy: October 7, 1925; death same day.

Referred by Dr. T. B. Ackerly, of Glastonburg, Connecticut, with the diagnosis of a brain tumor.

Complaints. Headache and vomiting.

Family history and past history. Negative.

Present illness. Six months ago the patient was awakened with a very severe headache which started in the frontal region and then spread over the whole head. He was nauseated and vomited. Since that time he has had four exactly similar attacks. In each instance, except the last one, headache lasted only a day. In the last attack it persisted for four days. There was slight stiffness in his neck. Vision was blurred during the attacks. At no time was consciousness lost. There have been no convulsions and no dizziness.

Physical and neurological examinations. Patient is a well nourished, well developed white man. There is symmetrical papilloedema of both discs, measuring about three diopters. There are no other positive neurological findings. The reflexes are unchanged.

X-ray of the head is negative.

Neurological diagnosis. Unlocalizable tumor of the brain.

Ventriculography, October 6, 1925. Twenty-five cubic centimeters of fluid was removed from the right ventricle and an equal amount of air injected. The right ventricle was perhaps somewhat enlarged. The third ventricle was pushed toward the right side. No air reached the left lateral ventricle; there must therefore, be a tumor in the left frontal region.

Operation, October 7, 1925. Under ether anesthesia a small bone flap was turned down in the left frontal region, using the hypophyseal approach with the concealed incision. Another opening was made over the right anterior horn in order to tap this ventricle which did not communicate with the left side. When the left hemisphere was exposed a needle was inserted and entered a greatly enlarged lateral ventricle. This finding indicated that we were dealing with an intraventricular tumor, for a large tumor in the frontal lobe would have obliterated this ventricle. It was evident also from the ventriculographic findings that the tumor must be at the foramen of Monro. A nasal dilator was passed through the cortex and after evacuating the fluid a reddish-brown mass was seen on the mesial wall

of the ventricle and covering the foramen of Monro. The tumor was reddish-pink, nodular, firm and was seen to be pedunculated. The pedicle was attached to the mesial wall of the ventricle. In order to remove it deliberately, resection of the frontal lobe was necessary; the entire anterior portion of the lobe (75 grams) was, therefore, excised. Very little bleeding resulted from removal of the lobe. The tumor was now in full view. The attachment of the tumor to the wall of the ventricle was dissected away and the tumor cut across at the foramen of Monro; it had protruded through this opening into the third ventricle. Its capsule was picked up and this portion easily delivered. The foramen of Monro was greatly enlarged measuring about 2 cm. vertically and 1.5 cm. in the anteroposterior direction. The tumor was clearly a pedunculated ependymal tumor which had arisen from the mesial wall of the lateral ventricle and had grown mesially into the third ventricle and laterally into the lateral ventricle. It weighed 9.59 grams.

During the removal of the nodule from the third ventricle a vein was torn in the wall of the foramen of Monro. From this a rather brisk hemorrhage resulted, but was suppressed by applying of packs of moist cotton and later a silver clip. The clip, however, was defective and tore the vessel, producing further bleeding which was finally controlled, though after a considerable loss of blood. Closure was made in the usual fashion, the bone flap being replaced.

His condition seemed good at the end of the operation, but a few hours later it was evident from his deepening coma that there had been intracranial bleeding. The wound was reopened and a large blood clot was found filling the lateral ventricle and the defect from which the frontal lobe had been removed. The clot was removed as far as it was possible to do so. The wound was again closed, but he became progressively worse and died during the night.

Microscopic note. The tumor presents a uniform picture of fairly closely packed, irregularly round, small vesicular nuclei

(Fig. 26) in a reticulum of fine connective tissue, which probably represents less than half of the tumor's bulk. The tumor is lined by a thin layer of more tightly compressed and rela-

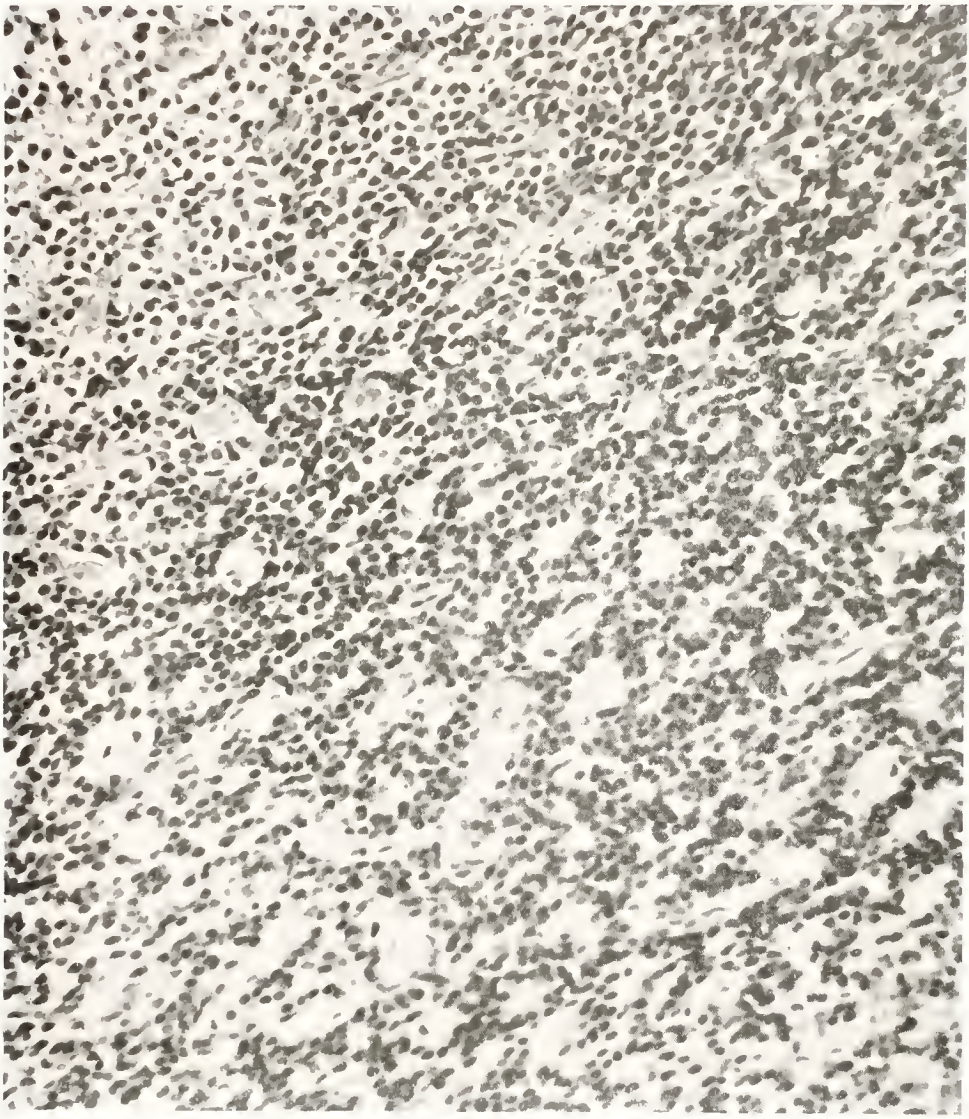

Fig. 26. Photomicrograph of tumor Case IV

tively acellular fibrous tissue. In places a low epithelial or flat endothelial layer is present. No neurogliar fibers are seen in the sections stained for glia.

Diagnosis. Ependymal (cellular) fibroma?

Case V

W. S. R. Age 38. Admitted: September 2, 1930. Total enucleation of tumor. Discharged: September 12, 1930.

Referred by Dr. Angus L. MacLean, Baltimore, to whom patient had been referred by Dr. Frank H. Hedges, of Frederick, Maryland, because of loss of vision.

Complaint. Failing vision.

Family history and past history. Negative.

Present illness. Fifteen months ago patient observed that his vision was blurred and that when driving an automobile stationary objects along side of the road seemed to jump in front of him. About this time he saw double, but this shortly passed away and has not again recurred. About three months later his vision began to improve without apparent cause and this improvement persisted for another three months, after which it began to fail rather rapidly and persistently. When attempting to read, the words would run together. At the onset of his visual disturbance sick-headaches developed; the pain was localized in the mid-sagittal and mid-frontal regions, and was quite severe. The attacks came on about once a week, and usually were associated with nausea and vomiting. In the last nine months his glasses have been changed several times, without any improvement resulting. His vision is now practically gone. He can detect lights with the right eye, and can only count fingers with the left.

There have been no convulsions; no paralyses, no mental changes, no disturbance of speech; in fact he has been entirely free of symptoms excepting those enumerated.

Physical and neurological examinations. The patient is a well nourished and well developed, normal appearing man. He does not appear ill. There is no sign of hemianopsia in the restricted vision of the left eye; the right eye is blind. Patient is unable to recognize colors. The visual acuity is 2/200. There is papilloedema of low grade in both discs. The vessels are somewhat full and tortuous; there are no hemorrhages. There are no extraocular palsies; no nystagmus. The audiome-

ter test shows a loss of hearing for high tones in both ears, much more marked in the right. Whether or not this bears any significance I am unable to say. The neurological examination otherwise is entirely negative.

X-rays of the head were negative.

Clinical impression. That the patient had a brain tumor was certain, but its localization from all of our examinations was impossible.

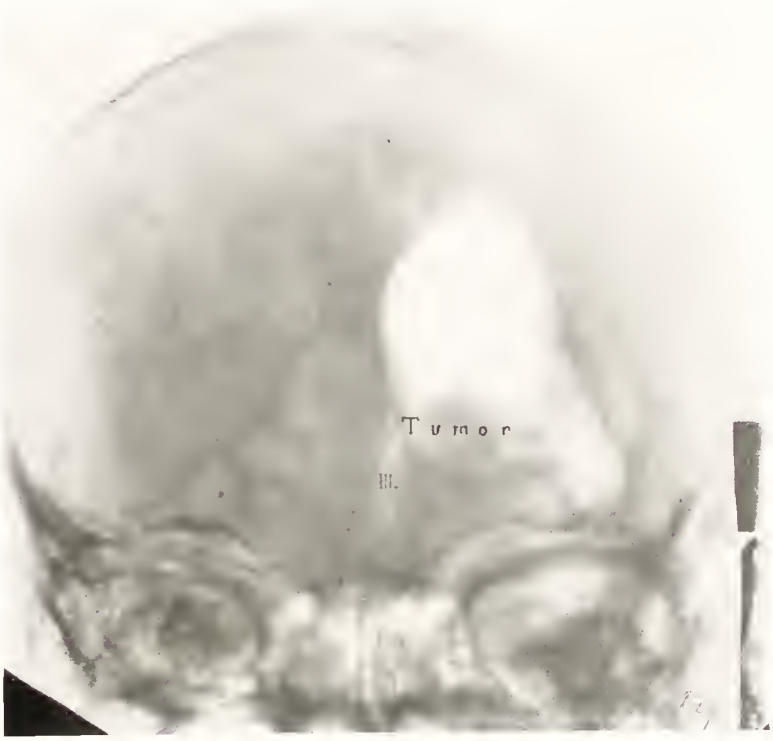

FIG. 27.1. Anteroposterior ventriculogram Case V, showing block at the foramen of Monro, also filling defect of the tumor.

Ventriculography was performed on September 3, 1930. The left ventricle was tapped first; there was tremendous hydrocephalus, fluid spurting under great pressure. One hundred cubic centimeters of fluid were removed and an equal amount of air injected. The whole ventricle was uniformly dilated; the third ventricle also filled, but only partially. There was, however, no dislocation of the ventricular system and none

of the air entered the right ventricle. There must, therefore, be a tumor at the foramen of Monro on the right side. The right ventricle was then injected, it was equally as large (Fig. 27.1). A large filling defect of the tumor is shown in the region of the foramen of Monro (Figs. 27.1 and 27*B*).

Operation, September 3, 1930. Under avertin anesthesia, a small (hypophyseal) bone flap was turned down in the right

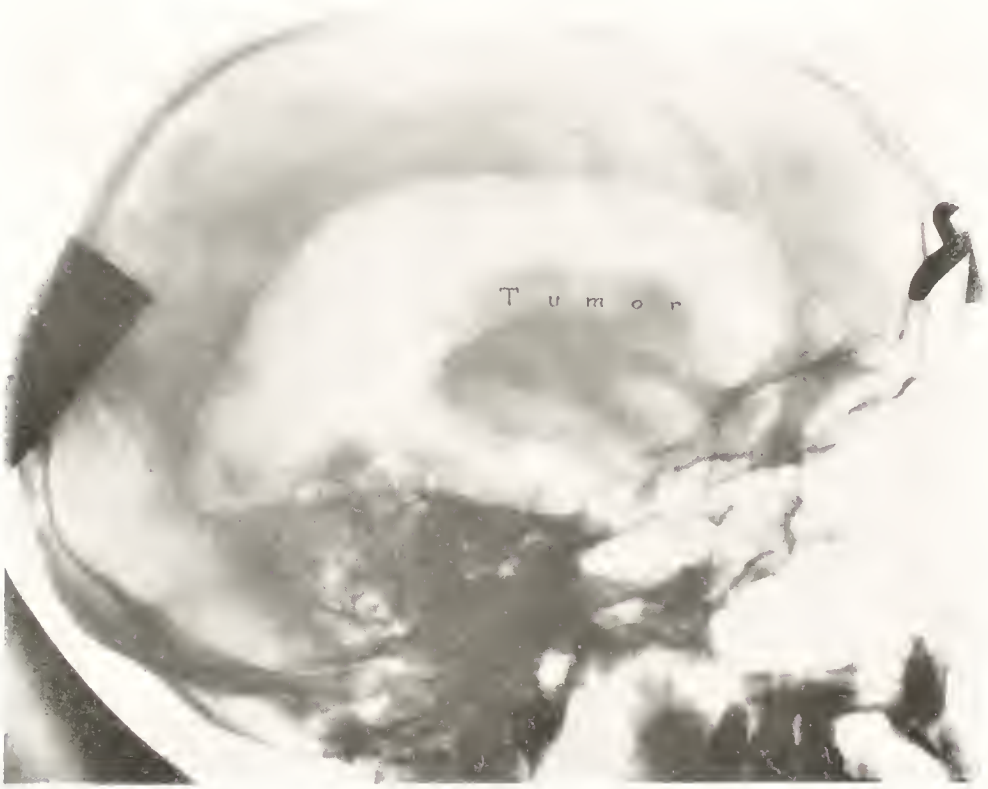

FIG. 27*B*. Lateral ventriculogram Case V. Note the tremendous enlargement of the ventricle and the filling defect of the tumor in its anterior third.

frontal region, using the concealed incision. The left ventricle was tapped through a perforator opening that had been made anteriorly on that side before beginning the cranial exposure. With pressure on the dura a large amount of air and fluid escaped and greatly reduced the intracranial pressure. After opening the dura the right lateral ventricle was tapped and

it was found to be of very great size and a large quantity of fluid could be forced from the needle by pressure on the frontal

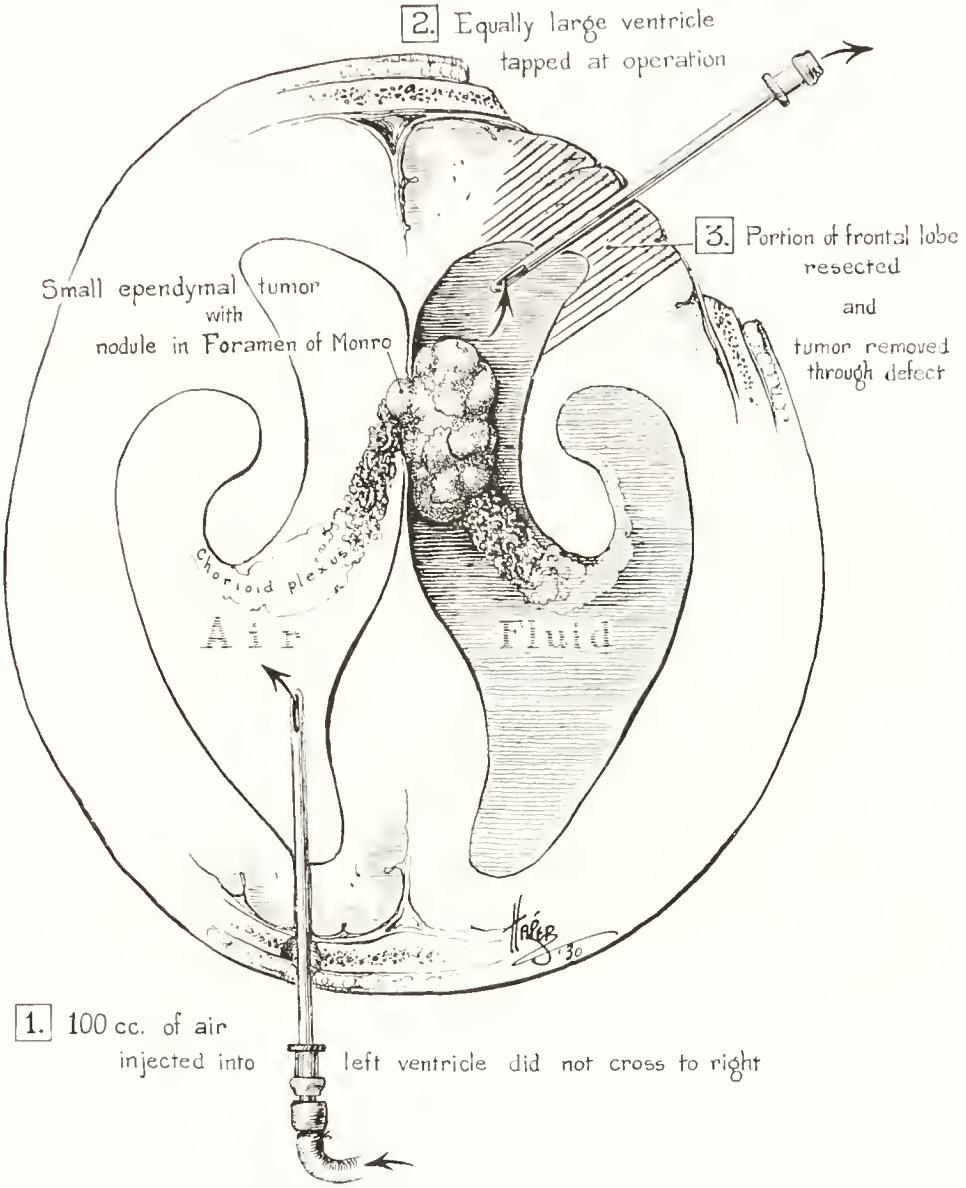

FIG. 28. Sketch showing position and general character of the tumor in Case V

lobe. A nasal dilator was then passed through the cortex into the anterior horn of the lateral ventricle where one could then see a pale grayish-white tumor with an irregular nodular sur-

face. The tumor was perhaps 4 cm. long and 2 cm. wide. At first the color of the tumor, and its seemingly insensible transition with the ventricular wall, made it appear that it might be infiltrating; but being hard and nodular this made an infiltrating tumor highly improbable. To be absolutely certain of its nature a "U" shaped area of frontal lobe was

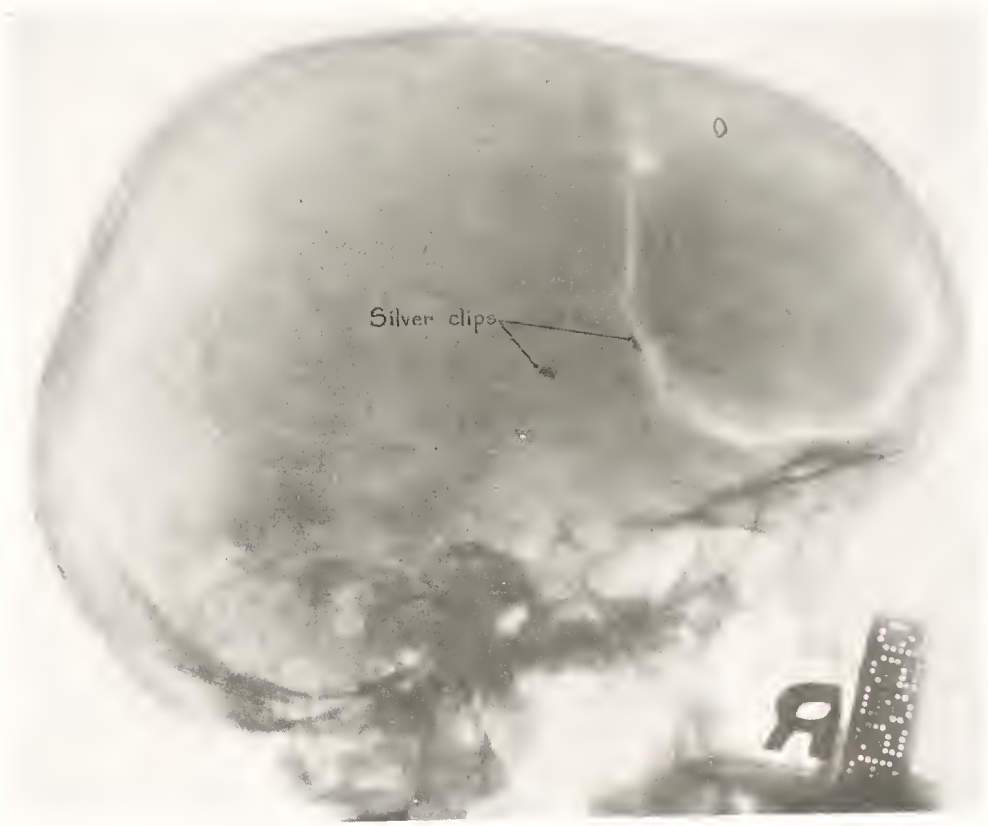

Fig. 29.1. The silver clips which were placed upon the blood vessels at the time of operation indicate the position of the tumor in the lateral view. The size and position of the operative approach are also indicated. (Case V.)

excised, thus permitting free access to the anterior part of the lateral ventricle. There remained, therefore, an area of brain tissue along the falx and another along the roof of the orbit. The tumor was now in full view (Fig. 28). By very cautious dissection it was soon found possible to dislodge the tumor entirely from the wall of the lateral ventricle. There were

three large veins which crossed upwards from the foramen of Monro along the mesial wall of the anterior horn of the ventricle; these were carefully avoided in dissecting the tumor and were not torn.

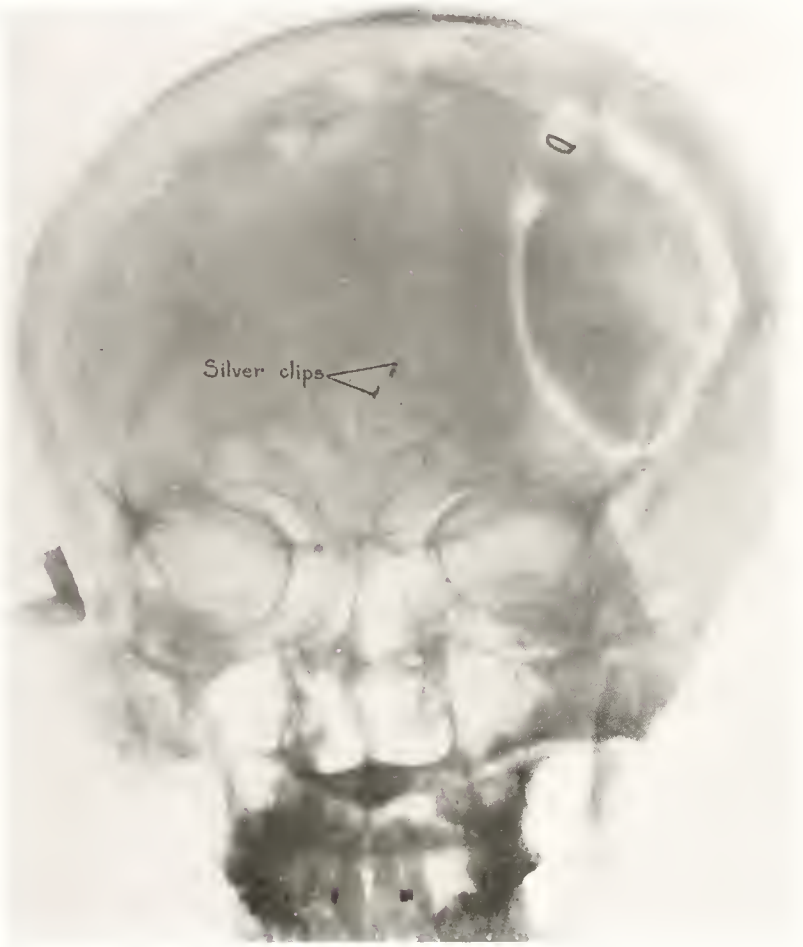

FIG. 29*B*. Silver clips show the position of the tumor in the anteroposterior view. (Case V.)

The tumor was now liberated everywhere except at the foramen of Monro where it could be seen protruding into and entirely filling this opening, and as the tumor was gradually withdrawn by gentle traction a small enlargement of the growth could be seen projecting into the third ventricle (Figs. 29*A* and 29*B*). No arterial or venous supply was disclosed in re-

moving the tumor, for there was no bleeding at any time. The tumor weighed 7 grams.

The wound was closed without drainage; the bone flap was replaced and wired.

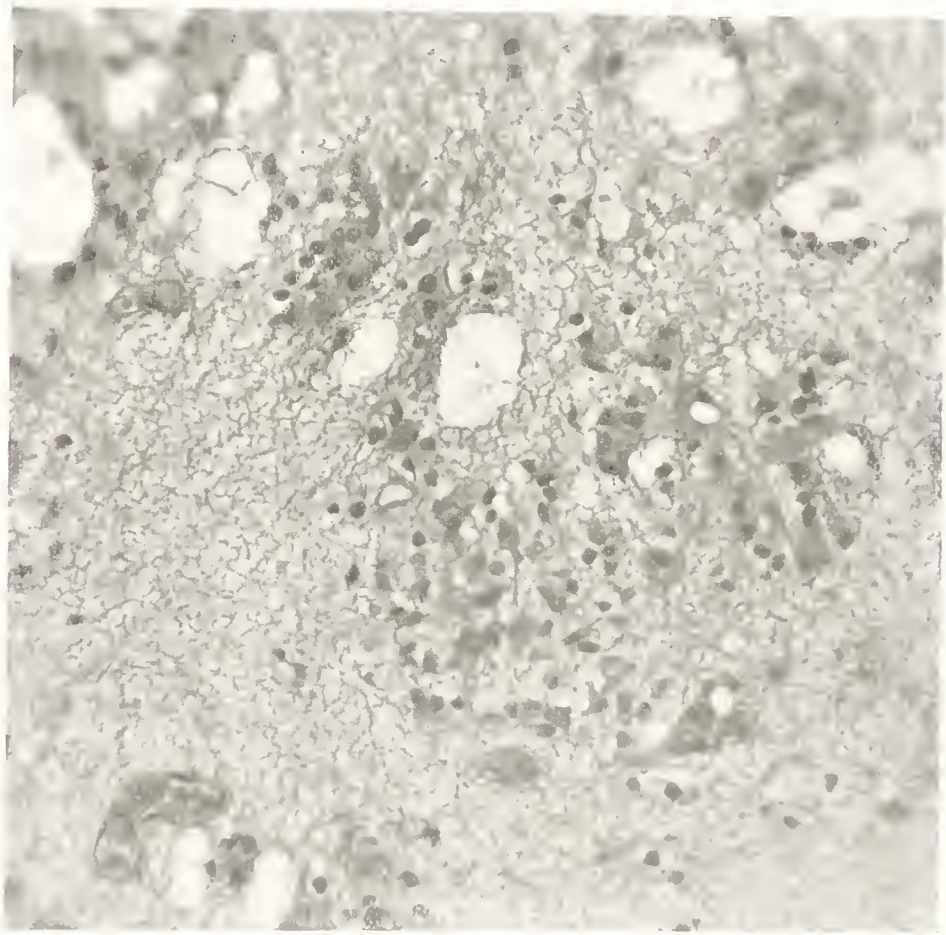

FIG. 30. Photomicrograph of tumor Case V

The patient made an uneventful recovery and was discharged from the hospital on September 12, 1930, ten days following the operation.

Microscopic note. The striking feature is a loose reticulum of fine fibrillar strands, with relatively few nuclei (Fig. 30). In places the nuclei which are irregularly round and vesicular are collected in little clusters, but nowhere closely packed. In

many fields under high power there will be only a few and at times no nuclei. There are many holes of varying size throughout the section; these are not lined by endothelium and appear to be merely cavities in the fine fibrillar network. A band of more compact fibrous tissue forms the capsule, which has no

FIG. 31. Photomicrograph (Case V) showing glial fibers in the tumor

visible epithelial lining. Glial fibers are in abundance and definitely form many parts of the basic fibrillar network (Fig. 31). In other areas the fibrillar base is not glial, but is apparently of connective tissue origin.

Is this tumor like that of Case IV except that the cellular element is less and the fibrous far greater?

Diagnosis. Ependymal glioma or fibroma.

Subsequent course. On January 5, 1931, four months after the operation his eyes were examined by Dr. MacLean, who found light perception in the right eye. The visual fields on the left were practically the same as before operation; there was no color perception; visual acuity had increased to 5/50. In other words, there was a slight but definite improvement in the vision.

When seen $1\frac{1}{2}$ years after operation his condition was practically unchanged. He has had three convulsions.

Case VI

R. A. Age 40. Admitted: May 31, 1931. Total enucleation of tumor. Discharged: June 25, 1931.

Complaints. (1) Knees give way; (2) poor vision; (3) headache; (4) mental changes.

Present illness. Patient was nervous on return from tuberculosis sanitarium four years ago; was irritable and jumpy. Severe headaches appeared about that time; they were bifrontal and radiated at times to the occiput. Each attack lasted four to five hours; vomiting with headaches was explosive and without nausea. One or two attacks of headache and vomiting occurred per month. Because they were so severe he had to give up his position as accountant. For six months thereafter he tried other work in another city but could not keep at it and for the past two years has made no attempt to work. The headaches gradually became more and more severe until now there are several attacks a week; they may be either frontal or occipital; his wife says they are more in the frontal region, and he says they are worse in the occiput. Eighteen months ago his left leg began to drag slightly and he has become generally weaker. Patient says that for the past three, or possibly four, years he has had difficulty in seeing with his left eye; for the past two years vision in the left eye has been practically lost; in the past six months there has been some diminution of vision in the right eye.

About a year ago patient began having convulsions; several times he was picked up on the street. He would frequently have as many as five or six, sometimes fifteen, attacks per day; these would last from a few seconds to ten minutes; they were clonic, and frequently rectal and bladder control were lost. Focal signs have not been observed. He also had many petit mal attacks, in which he would have a staring expression and might or might not fall.

During the past year patient has had marked personality changes; talks to himself a great deal; suggests committing suicide. He stares at people in a peculiar manner, so much so as to frighten them and causes his wife a great deal of embarrassment. He is very forgetful; pouts and cries with little provocation, frequently acts like a child. He has become very slovenly even to the point of urinating and defacting at any place. In explanation he says he has no control, which may or may not be true, but it sounds more in keeping with his other mental stigmata. There has been loss of libido for the past eighteen months. The patient has never had double vision before the loss of sight in the left eye; nor has there been a history of hemianopsia. He has nycturia, now only once or twice per night, but formerly was four or five times.

Examination. The patient is a fairly normal appearing, well nourished man, somewhat euphoric, but answers questions quite sensibly. He is, however, quite hazy on facts and history. Aside from the fact that he has had a shoulder girdle amputation following trauma, physical examination is negative.

Neurological examination. Positive findings are: left pupil does not react to light or accommodation; the left eye is practically blind, only light preception remaining. The field of vision in the right eye is normal except for a greatly enlarged blind spot (Fig. 32); visual acuity is 20/20. Papilloedema of three diopters is present on the right and two on the left. The veins are greatly enlarged and tortuous; the discs are entirely obliterated, but on the left there is a greater degree of pallor, doubtless in keeping with the atrophy of this nerve.

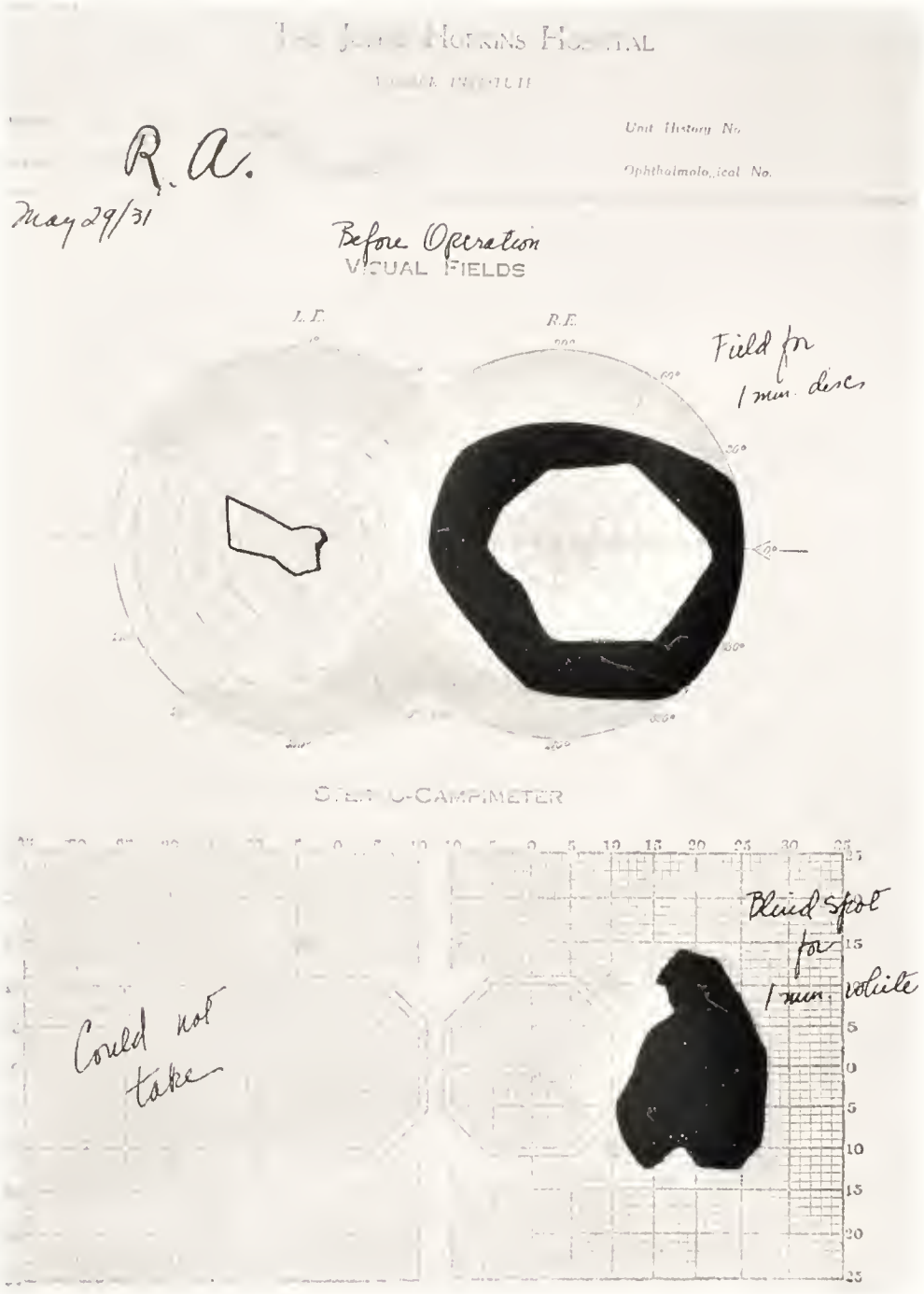

FIG. 32. Visual fields of Case VI before operation. They were unchanged at the time of discharge. Note the almost total loss of vision in the left eye, which probably is not dependent upon the tumor. Also note the large blind spot in the good eye.

There is general motor weakness which is possibly, but not definitely, greater in the left leg, but there are no changes in the reflexes to correlate with such findings. He walks without a limp. There is no spasticity, no clonus; negative Babinski on each side. There is no disturbance of any of the extraocular muscles and none of equilibrium.

Audiometer curves show some loss of high tones in both ears (Fig. 33).

X-ray of the chest shows an old healed tuberculosis of the right upper lobe.

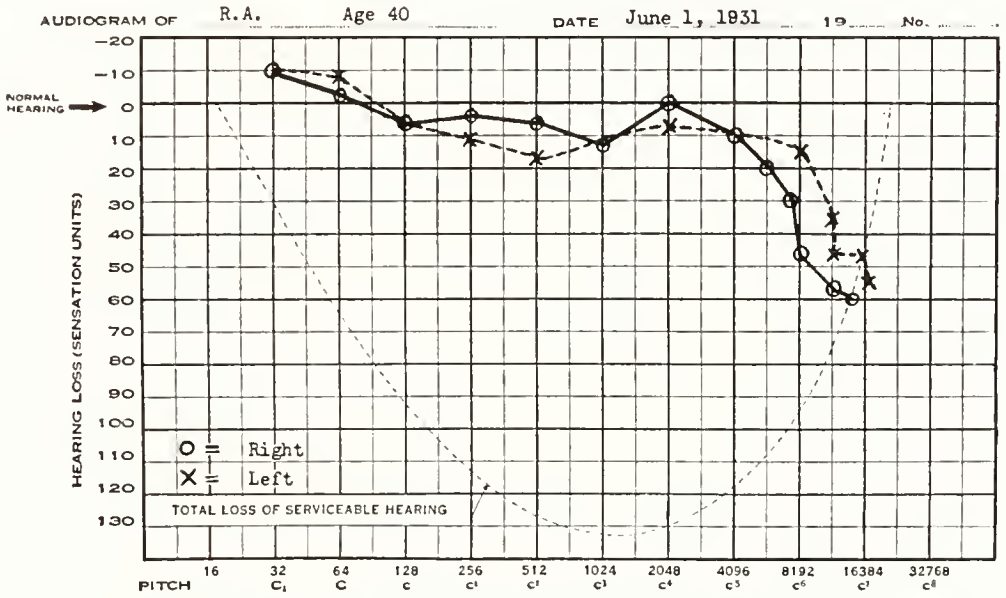

FIG. 33. Audiometer curves showing loss of hearing for high tones in both ears

X-ray of the head was negative.

Wassermann reaction was negative. Patient had had a history of possible chancre, but repeated Wassermann reactions have been taken and all are said to have been negative.

Impression. That the patient had an intracranial space occupying lesion of some type was clear; there was nothing to indicate its location. In view of the fact that he had been treated for tuberculosis in a sanitarium, and that there was roentgenographic evidence of an old tuberculosis of the apex

of one lung, I was inclined to suspect an intracranial tubercle. Repeated medical examinations, however, revealed no evidence of any active pulmonary process.

Ventriculography. The localization of the tumor was made solely by ventriculography. The right ventricle was tapped; fluid was under pressure; 150 cc. of fluid removed and an equal

FIG. 34.1. Lateral ventriculogram Case VI. Note the tremendous dilatation of the lateral ventricle and the filling defect of the tumor (arrows) in the region of the foramen of Monro.

amount of air injected. The left ventricle was not tapped. Air passed freely to the opposite lateral ventricle; both lateral ventricles were extremely large, but the right was considerably larger than the left. There was no sign of the third ventricle in any of the plates. No air reached the subarachnoid space. There was a beautiful filling defect in the right lateral ventricle in the region of the foramen of Monro; it could be seen in both

the lateral and anteroposterior plates (Figs. 34*A* and 34*B*), but was particularly well defined in the latter. It was a fairly round shadow about 1.5 x 1.5 cm. This is the third patient in whom a tumor of the third ventricle has been diagnosed when the air passed freely from one lateral ventricle to the other. Such a finding is, of course, possible only when there is a

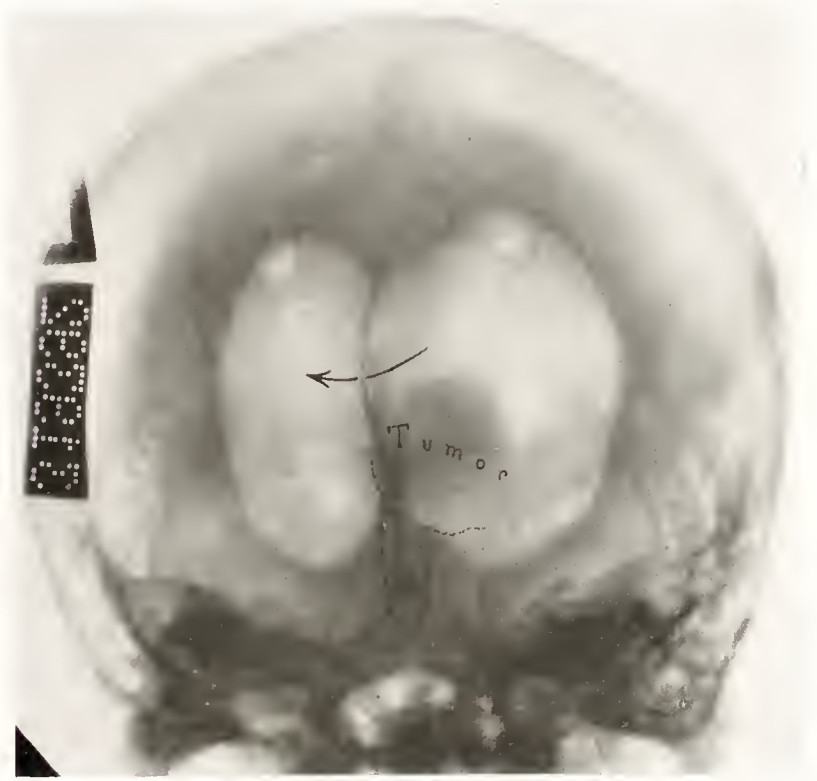

FIG. 34*B*. Anteroposterior ventriculogram showing filling defect of the tumor in the right lateral ventricle. Note the area of atrophy in the septum pellucidum (arrow). The dotted line below the ventriculographic shadows indicated the extent of the tumor into the third ventricle.

defect in the septum pellucidum (Fig. 34*B*) which results from very long continued pressure in a high grade of hydrocephalus.

Operation, June 2, 1931. Avertin anesthesia, supplemented with ether. A small bone flap was turned down in the right frontal region, using concealed incision; it is essentially the approach for hypophyseal tumors, though it does not extend quite so far forward. A ventricular needle was passed through

the dura and into the ventricle allowing air to escape freely. After opening the dura a nasal dilator was inserted into the

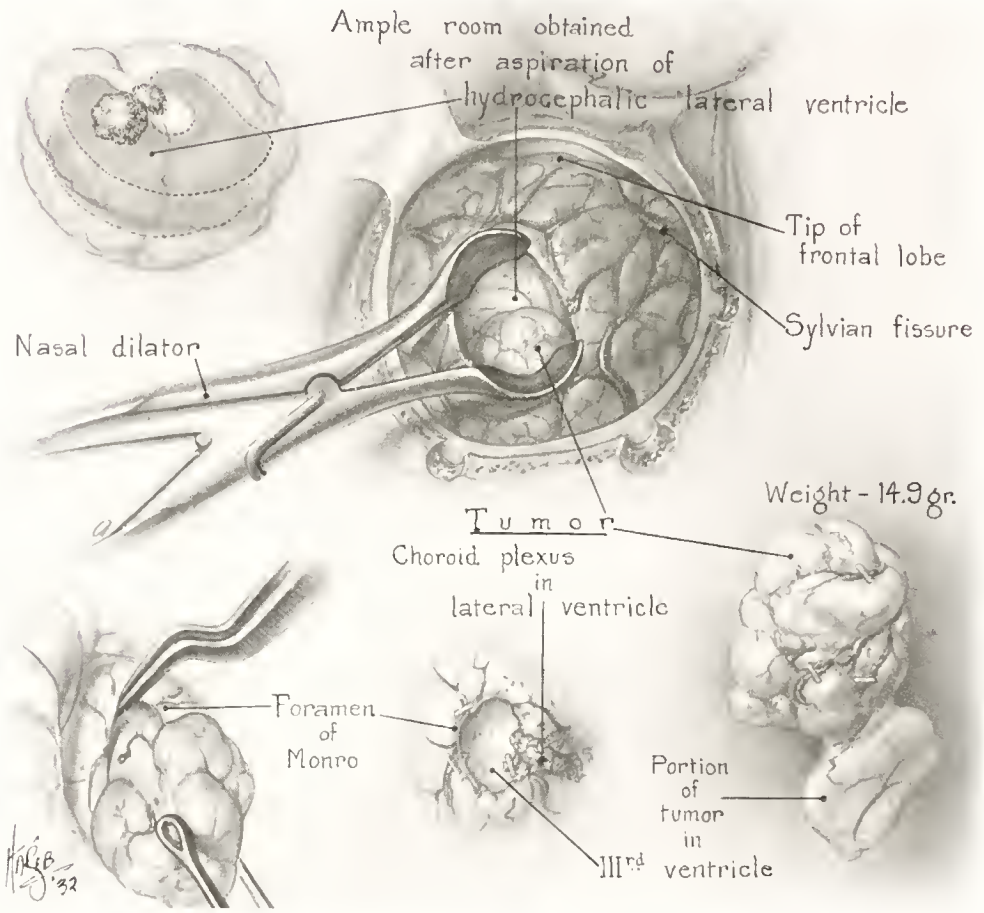

Fig. 35. Operative sketches of Case VI.

1. Disclosure of the tumor in the lateral ventricle through the separated nasal speculum.

2. A drawing of the tumor (upper left figure).

3. Tumor being drawn by gradual traction through the foramen of Monro (lower left figure).

4. Greatly enlarged foramen of Monro after delivery of the tumor. Note the clips on the vessels in the choroid plexus and veins in the wall of the foramen of Monro (lower central figure).

5. Sketch of tumor. The lower portion is flattened and is almost exactly the shadow of the third ventricle. The larger portion of the tumor beyond the constricted neck lies within the lateral ventricle and is responsible for the filling defect which is shown in the ventriculograms (lower right figure).

anterior horn of the ventricle. One could then see a white, hard nodular tumor projecting from the inner wall of the lateral

ventricle (Fig. 35); when it was touched with forceps it could be moved quite freely, thus indicting that it was a pedunculated growth. An area of frontal lobe of horse-shoe shape was removed; there still remained a good margin of cerebral tissue along the roof of the orbit and the falx. The width of brain tissue removed was about 3 cm., but it was ample because of the large ventricle. The raw surfaces of the brain were protected by pieces of cotton and with gentle retraction a good exposure of the tumor was possible. The tumor came directly out of the foramen of Monro which was greatly enlarged, perhaps to six or eight times its normal size, by the bulging mass. After passing through the margin of the foramen of Monro the tumor again bulged, filling the anterior part of the third ventricle. The anterior margin of the foramen of Monro was split in order to facilitate the dissection of the tumor. The portion of the tumor within the third ventricle was only about 1 cm. wide. The circumference of the tumor within the third ventricle was about as large as that of a twenty-five cent piece. It was necessary to strip the tumor very gently because of several veins which extended to the margins of the foramen of Monro; three of these were cut and thrombosed with the electro-cautery. They, however, were small branches. One of the large branches in the anterior horn of the lateral ventricle was gently stripped from the tumor to which it was but slightly attached; it coursed along the inferior surface of the tumor and along the inferior margins of the foramen of Monro and was directed toward the small vein of Galen. Over the superior surface of the tumor the tela choroidea of the roof of the third ventricle was attached but could be separated without bleeding. There were no other attachments except under the right foramen of Monro, and in stripping at this point a large vein was torn. This was promptly packed, later caught with a clamp and thrombosed with the electro-cautery. One could now see through the other foramen of Monro which was also quite large, but smaller than the right. After stopping this bleeding the wound was entirely dry. One could then see three open-

ings in the septum pellucidum; they were well back of the foramen of Monro. It was through these defects that the air passed freely from the right to the left lateral ventricle.

The weight of the tumor was 14.9 grams.

The dura was tightly closed; the bone flap wired in place. The galea and skin were closed with interrupted sutures of silk.

FIG. 36. Photograph of patient Case VI after operation

Microscopic note. Specimen has been lost.

Post-operative course. The post-operative course was uneventful, no motor, sensory, mental or other changes resulting from the operative procedure.

Subsequent course. Letter from his wife March 15, 1932,

states that patient has had two epileptic seizures. "Mr. A. (Fig. 36) has made rapid progress in many ways. He weighs 172 pounds: seems normal in every way, except that he still retains some of the strange ideas he had while at the hospital, and I cannot convince him differently." He has had three convulsions to date (September 1, 1933).

Case VII

L. D. Age 19. Admitted: August 3, 1930. Total nucleation of tumor. Discharged: August 25, 1930.

Referred by Dr. L. P. Martin, Marksville, North Carolina.

Complaints. Headache; convulsions.

Family history and past history. Negative.

Present illness. Began 18 months ago with a convulsion that involved both arms and legs. Her head was pulled backward; she bit her tongue; there was no loss of sphincter control. The attack lasted about ten minutes; she was totally unconscious; had no recollection of an aura. A month later she had a second attack during the night, and during the next year they occurred at intervals of three or four weeks but for the past four months have been absent. The patient states that she had severe headaches in the occipital region for three or four months before the time of her first convulsion. They always begin suddenly in the occipital region, reach the maximum intensity in a few moments and last for ten to thirty minutes, after which she is well except for exhaustion and nervousness. These headaches may occur every day for a week and then disappear for a few weeks. The occipital headaches are intensified after her convulsions. Recently they have become much more frequent, and on some days there have been as many as ten attacks. Vomiting occurs with most of the headaches and relieves them.

For the past six months patient has been unable to read because the letters run together and because attempts to read produce headache. There has been blurring of vision, but no diplopia. For the past month patient has felt a little unsteady

on her feet, but she does not think there was actual staggering. There has been some dizziness which is brought on by movement and disappears when she is quiet. At times she must stand still for a few moments because everything gets dark in front of her; she says she cannot see at all during these spells. Because the occipital headaches have been so much more severe and there has been so much vomiting and dizziness,

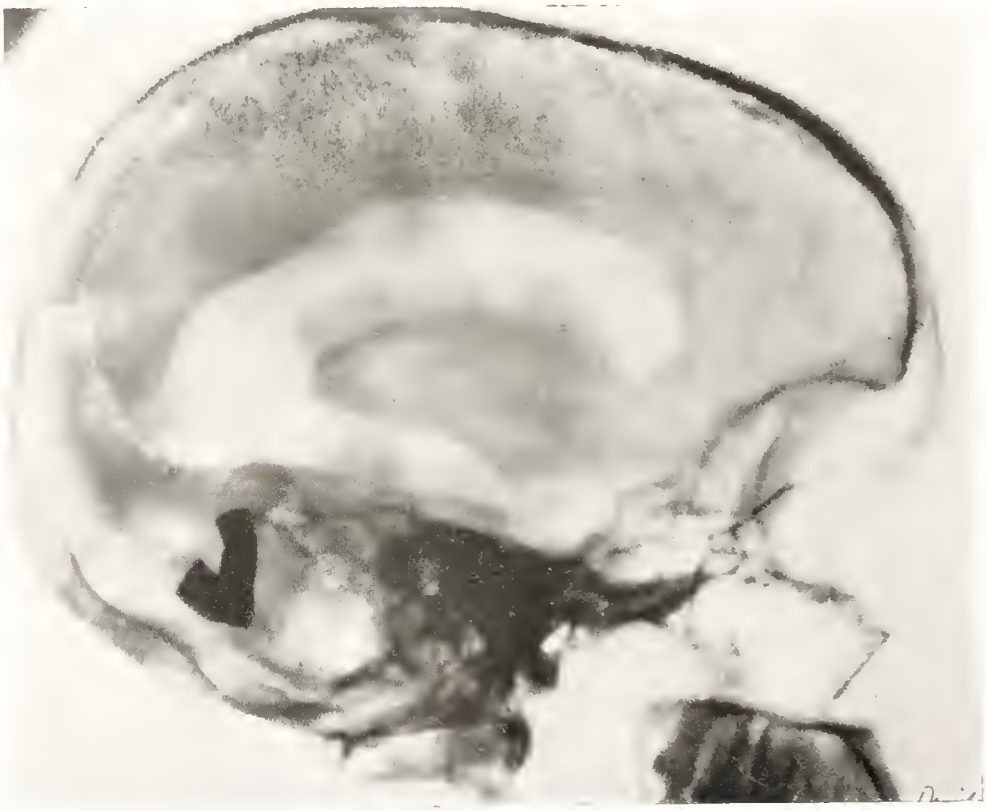

FIG. 37.1. Ventriculogram of Case VII. Normal left lateral ventricle

she has felt exhausted and has remained in bed for the past week. There have been no motor or sensory disturbances following the convulsions, nor at any other time. It is impossible to elicit any evidence of a localizing character in the attacks.

Physical and neurological examination. Patient is a well nourished young woman in good physical condition. Her

mental reactions are normal. There is a moderate degree of papilloedema in both eyegrounds. The margins of the discs are obscured; the veins are large and tortuous; there are no hemorrhages. There are no positive findings in the examination of the cranial nerves or the regions of the brain. The reflexes are normal. Blood pressure 124/66. It is worthy of

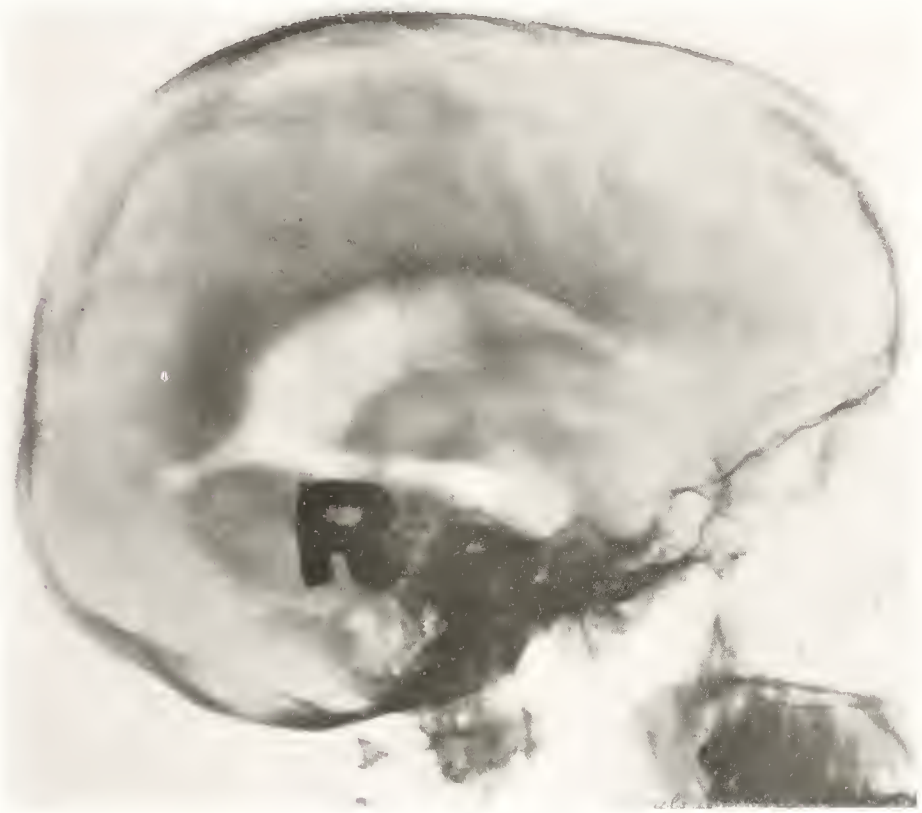

FIG. 37*B*. Ventriculogram of Case VII. Right lateral ventricle tapering at the anterior horn in the region of the tumor.

note that eight months ago she had a small tumor removed from her leg; it was diagnosed as a blood tumor. There has been no recurrence. Apparently no sections of the tumor were made.

X-rays of the head are negative.

Audiometer test is negative.

Ventriculography. In view of the negative findings the

ventricular system was injected with air August 8, 1930 (Figs. 37*A*, 37*B* and 37*C*). The left ventricle was first tapped and fluid spurted under tremendous pressure; 20 cc. of fluid were removed and an equal amount of air injected. The entire ventricular system was dislocated to the left; the third ventricle

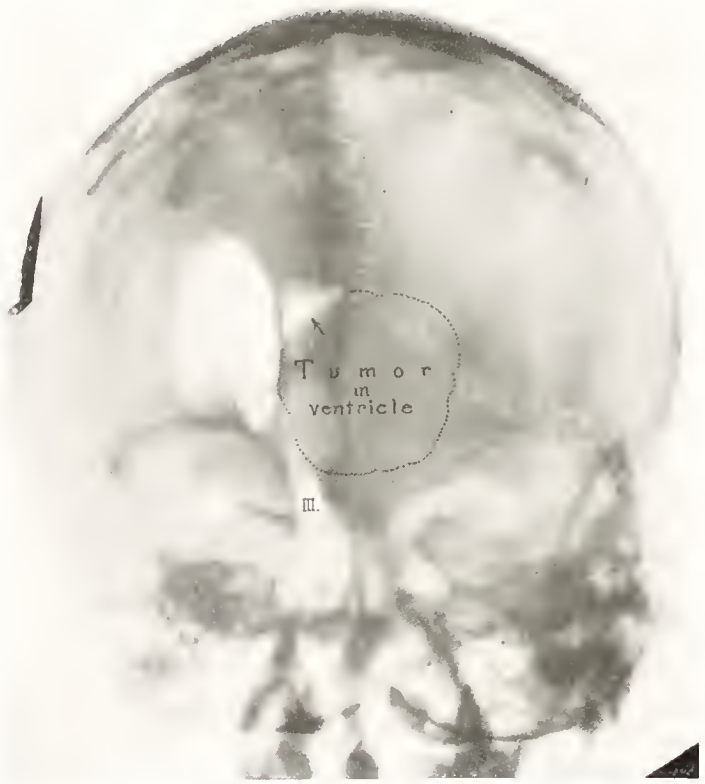

FIG. 37*C*. Ventriculogram of Case VII. Anteroposterior view showing dislocation of the ventricular system to the left and the small triangular remains of the right lateral ventricle which was cut away by the tumor.

was oblique (Fig. 37*C*). There was a large filling defect in the anterior horn of the right lateral ventricle (Fig. 37*C*).

Operation, August 8, 1930. The patient was immediately prepared for operation which was performed under *avertin* anesthesia. A large bone flap was turned down over the right frontal and parietal regions. The tumor had not reached the surface of the hemisphere, but there was distinct pallor and

softening of all of the frontal convolutions. Moreover, all of the fluid had been expressed (by an underlying tumor) from the subarachnoid space over the frontal lobe. The ventricular needle was inserted into the frontal region and met a firm resistance at a depth of 3 cm. below the surface; the tumor felt very hard. An incision was made through the cortex; the surface of the tumor gave every indication of encapsulation

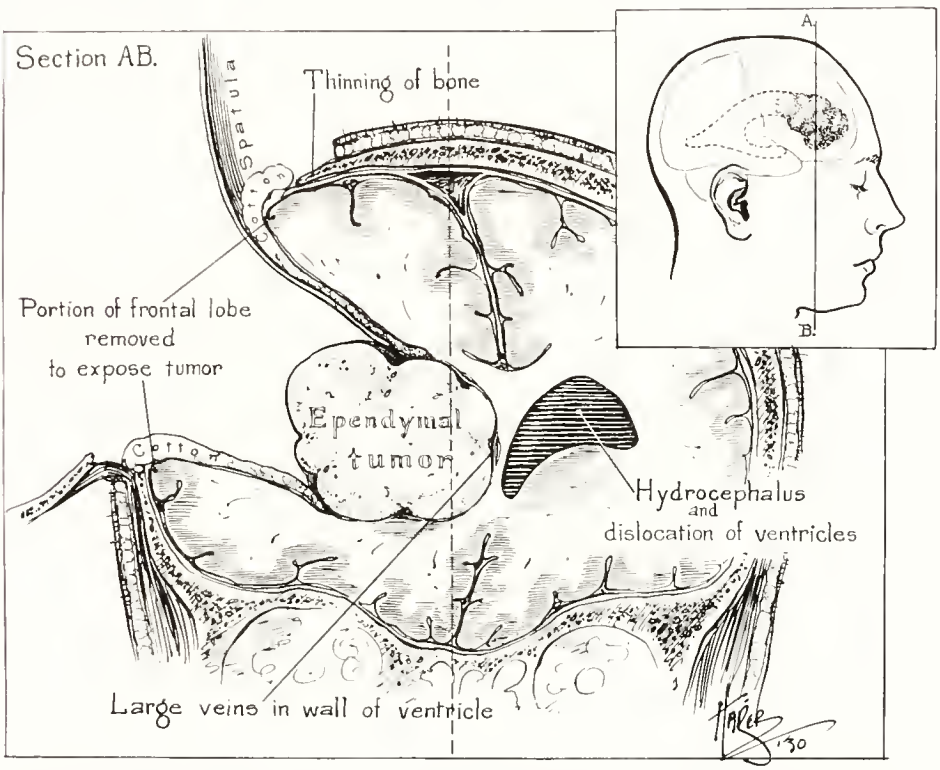

FIG. 38. Sketch from operative findings showing position of the tumor (inset) and the operative approach through the defect in the frontal lobe. (Case VII.)

(Fig. 38). The frontal lobe was then excised along a line just in front of the Rolandic area. The surface of the tumor was then in full view. The tumor extended to the anteriormost part of the cranial chamber. It was lying just above the roof of the orbit, but there was a definite layer of cerebral tissue interposed between the tumor and the orbital roof. The tumor was enucleated with the finger. There was quite a spurt of

blood which was controlled by packing with moist cotton, after which the source of bleeding was seen to be a large vein in the mesial wall of the lateral ventricle and in the region of the foramen of Monro. A clip was placed upon it; two other small veins were also clipped. The tumor arose from the wall of the ventricle just above the foramen of Monro (septum pellucidum). It was a beautiful, nodular, reddish tumor and was exceedingly hard and noncompressible. It looked very much like a dural endothelioma. A number of small, oozing

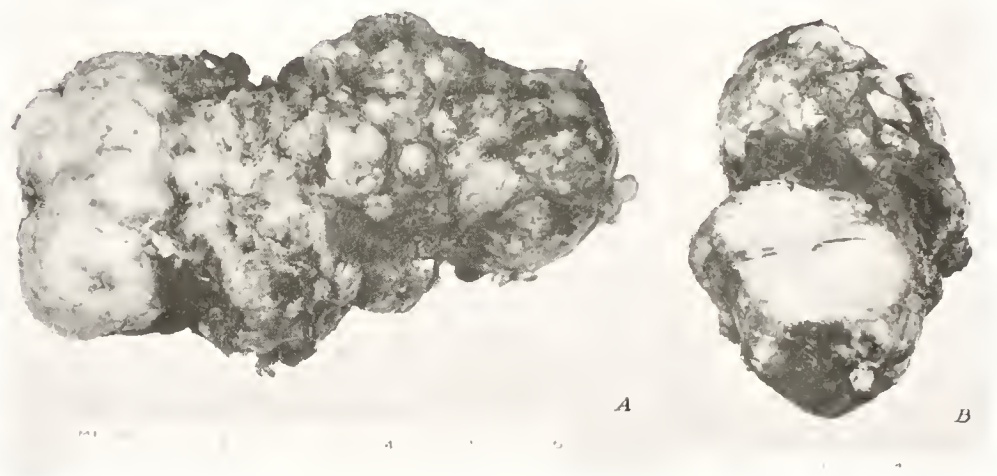

FIG. 39. *A*. Surface view of tumor in Case VII. *B*. Cut surface of tumor showing its hard fibrous character.

vessels were sealed with the electric cautery. The wound was dry when closure was begun. One now had a splendid view of the anterior portion of the right lateral ventricle and could see the choroid plexus passing through the foramen of Monro to the third ventricle.

The dura was closed tightly and the bone flap wired in place. The tumor weighed 47.7 grams (Fig. 39, *A* and *B*). It arose from the ependymal lining of the mesial wall of the lateral ventricle.

The patient made an uneventful recovery and was discharged August 25, 1930, seventeen days after the operation. She has

since been well (Fig. 40). There have been no mental or physical effects from the removal of the tumor or the right frontal lobe, and no convulsions.

Microscopic note. The tumor is made up of connective tissue of varying density; in places it has a loose areolar character with sparse cellular elements, but in far greater amount it is

FIG. 40. Photograph of patient taken two years after the operation

densely packed (Fig. 41) and frequently in whorls like a dural endothelioma. The cellular element varies in amount. In many places it predominates and little connective tissue can be seen. In other places there are few cells in a field. The nuclei are small, round, and oval, not unlike those in the preceding tumors. Many areas of extensive necrosis are present. A thin bed of parallel connective tissue fibers form

the capsule—no epithelial lining is seen and there are no epithelial cells throughout the tumor.

In one section the tumor is made up of myriads of small vessels with hyaline walls. Here a diagnosis of angioma might

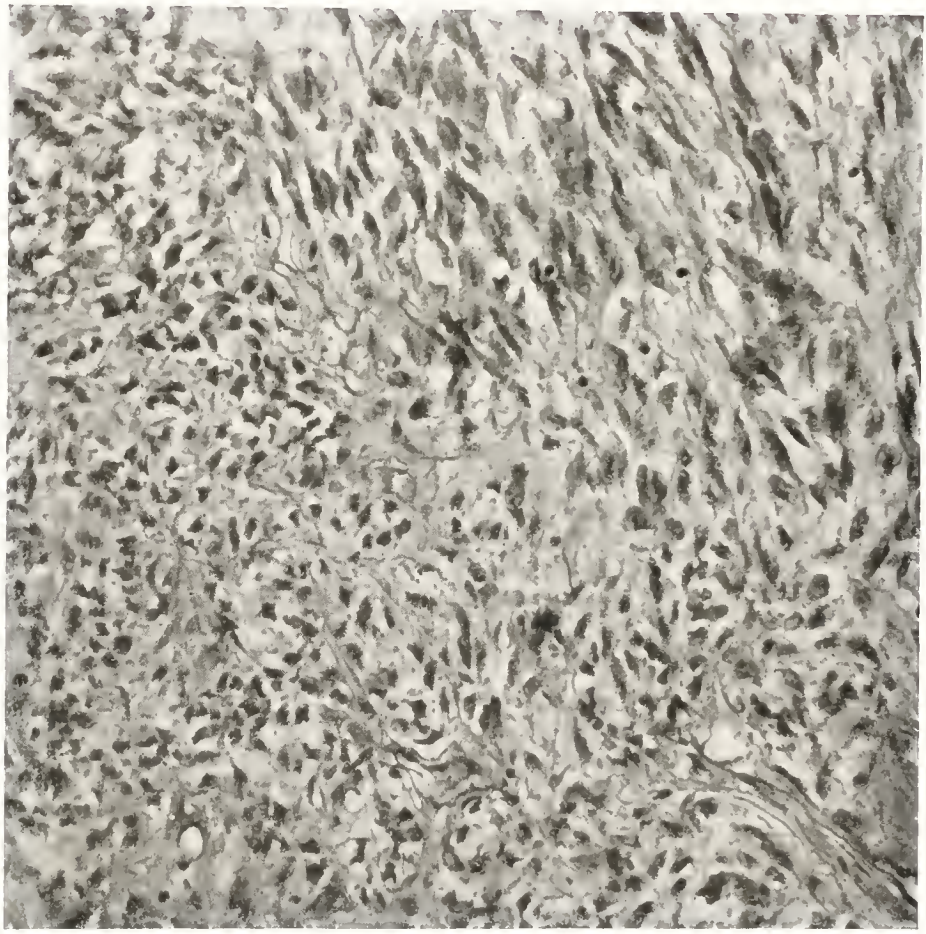

Fig. 41. Photomicrograph of tumor Case VII

well be made. There are many areas of colloid. Occasional tiny isolated patches of glial fibers are seen.

Diagnosis. Ependymal fibroma.

Case VIII

B. W. Age 33. Admitted: June 17, 1928. Total enucleation of tumor. Discharged: July 7, 1928.

Complaints. Blurring vision, headache, backache and numbness of right arm and leg.

Family history and past history. Negative.

Present illness. The patient dates his trouble to nine years ago (1919) when he began having headaches in the occipital region. He also had generalized weakness and shaking of the knees; he thought he was nervous. However, he was not greatly disturbed by his symptoms for he married the following year.

About four years ago (1924) he noticed the loss of hearing in the left ear; there were "thumping" noises in his right ear. These sounds would not be heard when he was quiet, but became marked when he heard some sound. The physician who treated him for his ears made an ophthalmoscopic examination of his eyes and told him there was evidence of intracranial pressure. Patient then noticed rapidly progressive loss of vision. He says that at times everything became blurred before his eyes and straight lines appeared crooked. He was sent to a neurological surgeon, who in 1927 did a right subtemporal decompression. During the following ten months his condition remained very much the same. At the present time (1928) he complains of severe headache and an occasional feeling of numbness and tingling in the right leg and arm. At times there is dizziness, and he walks as though he were drunk.

The *general physical examination* is entirely negative. The patient is a fairly well developed man of average size, well nourished; his mental status is essentially normal.

Neurological examination. There is marked diminution of vision in the right eye, less in the left; visual acuity in the former was 5/70, in the latter 20/50. Perimetric examination showed marked reduction in the fields of vision of both eyes, considerably more in the right. There is much greater loss of vision in the right nasal than the right temporal field, although there is no hemianopsia. There is also somewhat greater loss of vision in the left nasal than the left temporal field. Any suggestion of hemianopsia would, therefore, be of

binasal type, but doubtless the fields indicate only severe loss of vision from intracranial pressure. There is papilloedema of low grade in each fundus; the retinal veins are full and tortuous; there is an outstanding pallor of atrophy at the margins of the discs. There is almost total deafness of the left ear. The audiometer test shows practically loss for all tones up to 4096; hearing 8192, 10,000 to 15,000 is about normal. It is difficult to conceive of any relationship between his tumor and this unusual loss of only the low tones.

Except for the above findings there is no objective impairment of function in any of the remaining nerves or the regions of the brain. Reflexes are entirely negative. The Wassermann reaction from the blood is negative. X-ray of the head is negative.

Impression. One could only be certain that there was an intracranial tumor; there was not the slightest suggestion of its localization. The only objective finding that might have been interpreted as a localizing sign was deafness; the localization of a tumor on this finding would have led us far astray.

Ventriculography was performed June 19, 1928. The right ventricle was first tapped; fluid spurted under high pressure. Seventy-five cubic centimeters of ventricular fluid was removed and an equal amount of air was injected. A slight amount reached the third ventricle but none passed into the left lateral ventricle. No filling defect or other deformity of the ventricle was evident. In order to learn the findings of the opposite ventricle, air was introduced into the posterior horn of this ventricle; only 30 cc. of fluid could be obtained from this ventricle and an equal amount of air was injected. In the ventriculogram of this side it was seen that the ventricle ended abruptly about its middle, no air passing beyond. The anteroposterior view showed a beautiful filling defect in the left lateral ventricle and the two ventricles to be widely separated, each being dislocated from the midline. The shape of the left lateral ventricle (anteroposterior view) was greatly changed by the tumor within is interior.

Operation, June 19, 1928. After interpretation of the ventriculograms the patient was immediately prepared for operation. Before the anesthetic was given air was released from each ventricle separately because these cavities did not communicate. Under rectal ether-anesthesia a cranial exploration was made in the left frontal region, the concealed incision being used. The incision in the skin and bone extended almost to the midline. The left hemisphere was then gently retracted. The corpus callosum was soon brought into view and was found to be pale and thin from the underlying pressure. After division of this structure a reddish-brown tumor came into view. The split in the corpus callosum was extended and during this procedure the left lateral ventricle was opened. One could then see a large tumor filling the anterior half of the lateral ventricle. Much of the tumor dangled freely and could easily be moved back and forth. It was fairly firm and apparently a well encapsulated growth.

Its attachment was traced to the mesial wall of the ventricle, just anterior to the foramen of Monro, i.e., the septum pellucidum. A nodule of the tumor could be seen to project through the foramen of Monro into the third ventricle. It was not difficult to strip the tumor from its mural attachment, which was about as large as a 25-cent piece. In doing so the tumor was broken off at the foramen of Monro. It was then considered advisable to excise the area of the septum pellucidum from which the attachment of the tumor had been stripped. This excision allowed a full exposure of the anterior part of the right lateral ventricle. The wound now being quite dry, there was no difficulty in shelling out with a long dissector the small nodule of tumor that projected into the third ventricle. As there were no attachments, it was merely necessary to dislocate the nodule from its bed. The wound was perfectly dry before closure was begun. The tumor weighed 18 grams. The dura was closed tightly; the bone flap replaced and wired; no drainage.

Postoperative and subsequent course. The patient had com-

plete paralysis of the right leg and arm following the operation: also a motor aphasia. The aphasia cleared up completely: the leg and arm to a large extent, though the patient still has a decided limp and some impairment of function in the arm and hand. The patient has had perhaps a dozen convulsions begin-

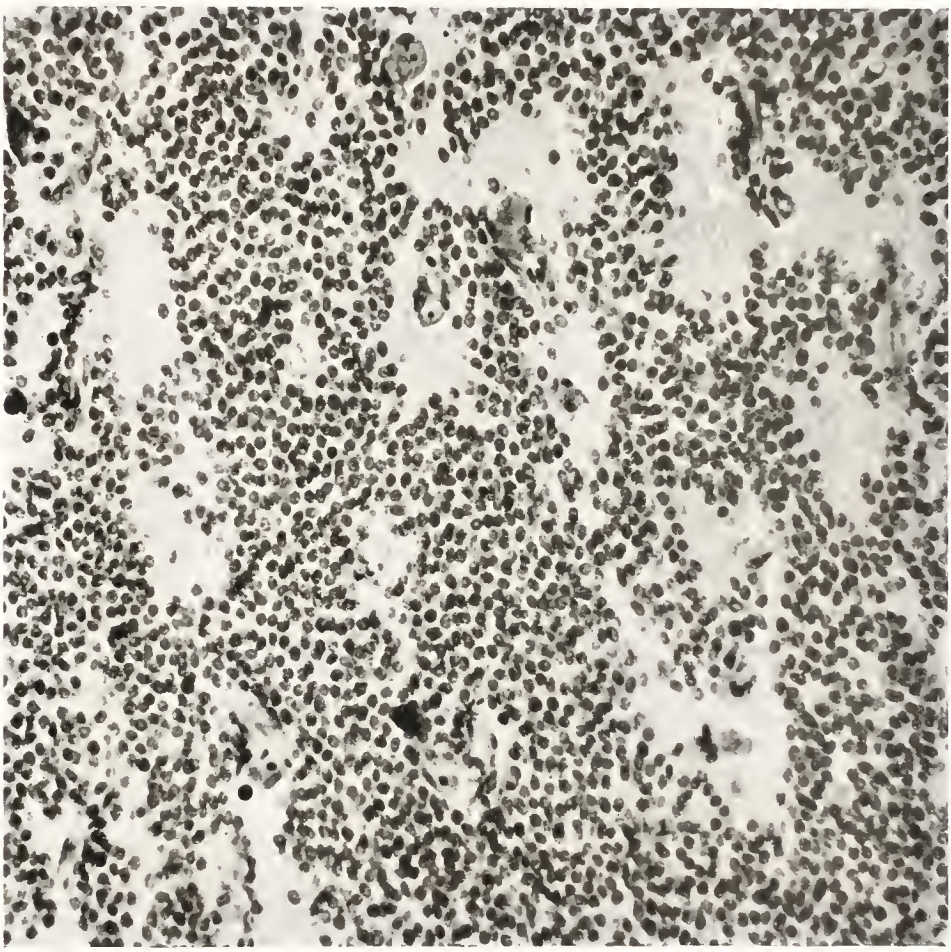

Fig. 42. Photomicrograph of Case VIII

ning in the right leg. His general condition is good; his old decompression is sunken; there have been no headaches.

Comment. It would unquestionably have been very much better to have made a hypophyseal approach with resection of the left frontal lobe than to have attempted the removal by

an exposure alongside the falx; one would then probably have avoided the motor injury. Since this operation we have also learned the great risk attending injuries to the anterior cerebral artery over the corpus callosum on the left side; we were fortunate in escaping this mishap, but it is a very greatly added risk.

Microscopic note. The section presents a fairly uniform picture of closely packed small cells (Fig. 42) with no arrangement. They are imbedded in a fibrous base which probably represents less than half of the tumor. The type of cells and the general picture of the tumor are almost identical with Case IV. There is a well defined capsule of connective tissue and a partial covering of flat ependymal cells. No glial fibers are seen.

Diagnosis. Ependymal (cellular) fibroma?

Case IX

E. E. S. Age 9. Admitted: January 16, 1931. Removal of tumor January 17, 1931. Death same day.

Referred by Dr. H. H. Ogburn, Greensboro, North Carolina, January 16, 1931, with the diagnosis of a brain tumor.

Complaints. Headaches and vomiting.

Family history and past history. Negative.

Present illness. Five months ago patient was nauseated and vomited, and immediately afterwards she had an excruciating left frontal headache. She was somewhat drowsy and after sleeping for several hours was again perfectly normal. She has had numerous similar attacks, as many as four or five a week, and at times she will vomit four or five times during an attack. Her headaches have always been on the left side. For the past six weeks the headaches have entirely disappeared, but she has had frequent vomiting spells and has been constantly more drowsy than usual. There have been no other complaints.

Physical and neurological examination. The patient is a normal appearing, well-nourished young girl of nine years. She has low grade of papilloedema in both discs; visual fields

and visual acuity are normal. There are no sensory, motor, or mental disturbances and no changes in the reflexes.

X-rays of the head show some convolutional atrophy, but are otherwise negative.

Clinical impression. Probably a tumor in the left cerebral hemisphere. For some reason my assistant, Dr. Briggs, suspected an intraventricular tumor, largely because of the negative findings.

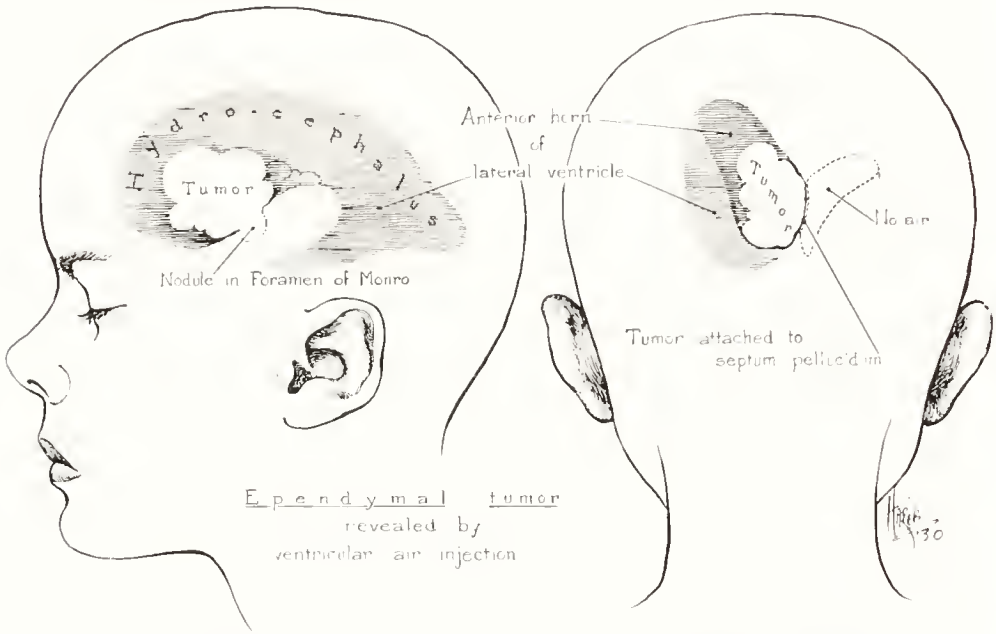

FIG. 43. Sketch showing the ventriculographic changes in Case IX

Ventriculography. January 17, 1931, the right ventricle was tapped and found to be of normal size and under only slight pressure. The left ventricle was then tapped and fluid spurted under tremendous pressure. The ventricle was very large; 75 cc. of fluid was removed and an equal amount of air injected. The air remained confined to this lateral ventricle, no air reaching the third or the opposite lateral ventricle. There was uniform dilatation of all parts of the left ventricle. When the ventriculograms were taken with the right side of the head down (that is in a position which allowed the air to

accumulate in the outer part of the left lateral ventricle) the outlines of the left lateral ventricle were of normal shape, though enlarged. When the left side of the head was down so that the air reached the mesial wall of the lateral ventricle, one could see a large circular filling-defect in the region of the foramen of Monro (Fig. 43). In the antero-posterior view the filling defect was even more beautifully disclosed (Fig. 43) on the mesial side of the ventricle.

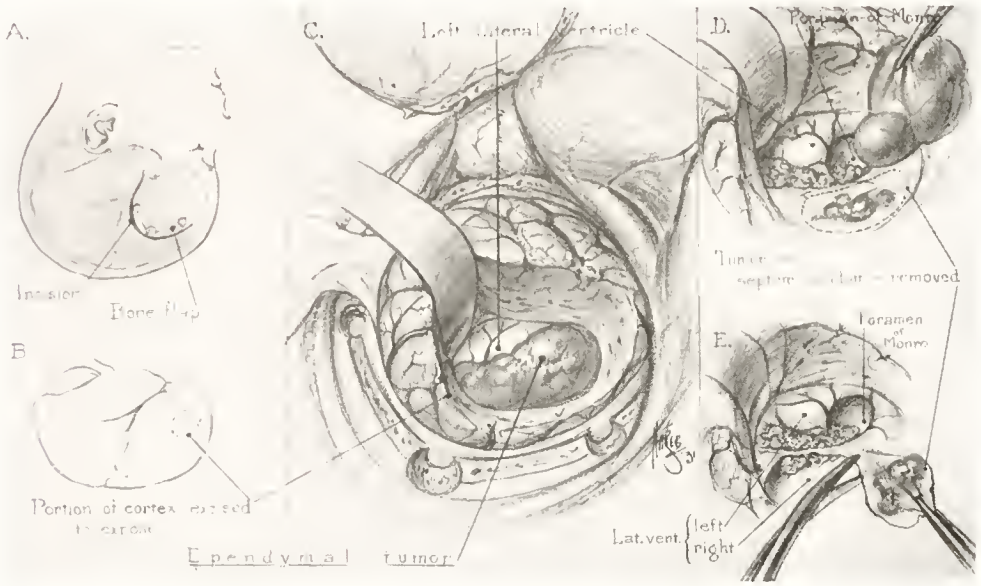

Fig. 44. Recapitulation of the operative story of tumor in Case IX

Operation. January 17, 1931; a small hypophyseal approach was made over the left frontal region, using the concealed incision (Fig. 44). Before opening the dura the ventricle was tapped and a fair amount of air escaped, but still the frontal lobe protruded moderately when the dura was opened. A "U" shaped area of frontal lobe was removed in order to expose the anterior part of the lateral ventricle. There remained, therefore, an area of brain tissue over the roof of the orbit and along the falx. The tumor was now in full view and it appeared to be very much larger than we had suspected, but the oval defect in the frontal lobe did not give us an exposure that was ade-

quate to extirpate the tumor. A complete resection of this part of the frontal lobe was then carried out. This resection was somewhat more bloody than usual, but the loss of blood gave us no apprehension. The bleeding was controlled before the extirpation of the tumor was begun. The growth was reddish-brown, soft and could easily be moved back and forth in the ventricle, which it almost filled. The tumor extended posteriorly into the body of the ventricle for quite a distance.

Fig. 45. Collected fragments of tumor in Case IX after operative removal

However, it was soon found that the tumor was pedunculated and attached to the mesial wall of the ventricle; most of this attachment was on the septum pellucidum, just anterior to the foramen of Monro. The tumor was so soft that it broke in fragments during the extirpation (Fig. 45). When the tumor had been removed from the lateral ventricle it was seen that there was still a small nodule firmly attached to the septum pellucidum at the raw area from which the main mass of tumor had been removed. Although there had been practically no

bleeding during the extirpation of the main mass of the tumor, the excision of this small nodule was accompanied by a brisk haemorrhage from the large veins that converged from the region of the foramen of Monro. These were thrombosed with the electro-cautery. The patient's pulse was quite weak and her blood pressure low at the end of the operation.

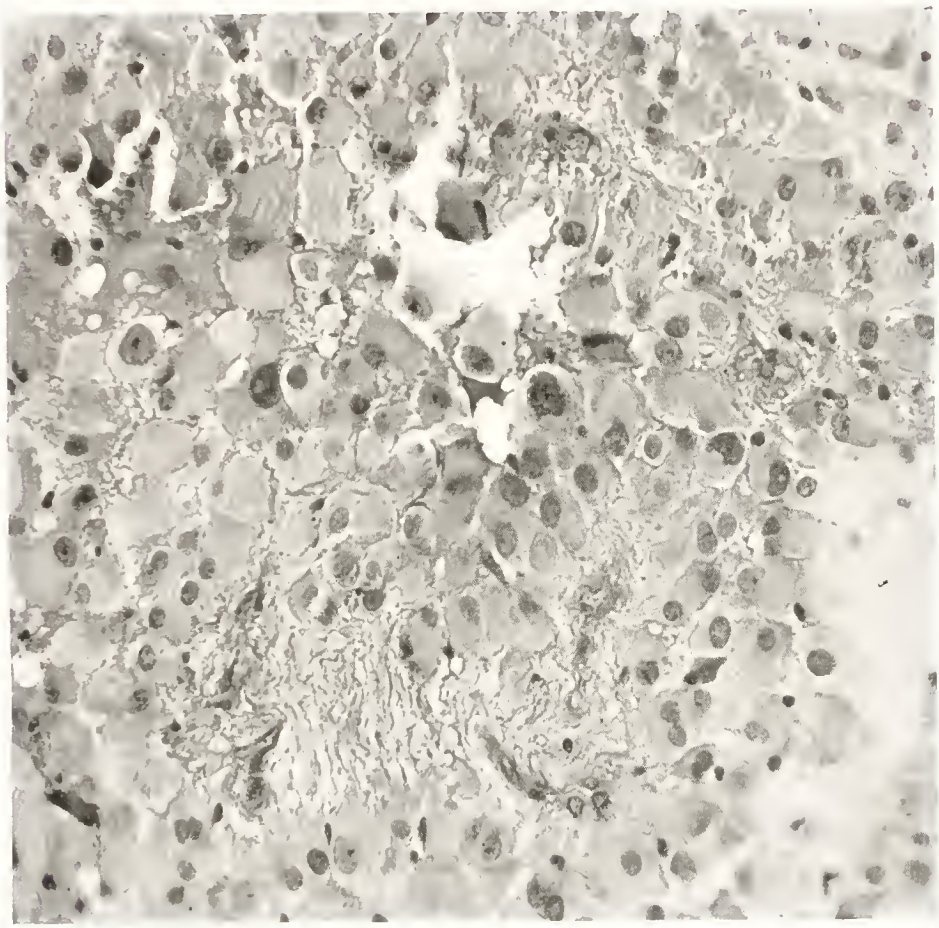

FIG. 46. Photomicrograph showing the large cells with excentric nuclei in Case IX

The wound was filled with Ringer's solution and closure conducted in the usual manner, the dura being closed tightly and the bone flap replaced. An intravenous injection of glucose was given at the end of the operation and caused immediate improvement in her general condition. However, this im-

provement was only transient and she soon showed signs of intracranial pressure, from which she died three hours later. Necropsy showed hemorrhage into the lateral ventricles and the frontal defect.

Weight of tumor 13 grams. Weight of brain tissue removed from the frontal lobe 66 grams.

Microscopic note. The tumor is exceedingly cellular and contains a minimum of stroma. The cells are very large and vary greatly in size and shape (Fig. 46). The cytoplasm stains deeply with eosin. The nucleus is large, vesicular, and of varying size; it is round or oval and contains a single large nucleolus. Nearly all the nuclei are excentrically placed and at the margin of the cell. At times there are two or three nuclei. In places cell boundaries are absent and one gets the impression of a syncytium. Occasionally a poorly formed acinus is seen and suggests a possible choroidal epithelium. However, there is nothing more to fortify this suggestion.

The microscopic appearance of its component cells closely resembles that of two cases recently reported in the tumors of the *third* ventricle (cases 6 and 7 of group II in that publication). Since the type of cells is unlike that of any known adult type, I was forced to conclude that the tumor was of embryonal epithelial origin. I can make no additional suggestion from the study of the sections in this case.

Diagnosis. Embryonal tumor of ependyma.

Case X

J. M. Age 14. Admitted: April 4, 1927. Total enucleation of tumor. Discharged: April 21, 1927.

A very poorly nourished girl of fourteen was referred by Dr. U. S. G. Ferrell, of Cairo, West Virginia.

Complaints. Failing vision, drowsiness and headaches.

Family and past histories are negative.

Present illness. Since the age of nine she has had bilious attacks with headaches; these occurred about three or four times a year, usually lasted about two hours. On August 7,

1925, 2½ years before admission, she complained of severe headache and lay down to rest. In a few moments she became delirious and remained so for four or five hours, after which she was given medicine by her physician, which quieted her and made her sleep. During the next day her physician noticed in the course of the routine examination that the vision in both eyes was greatly below normal, the loss being more prominent in the left eye. There had been no previous history of failing vision. A week later her tonsils and adenoids were removed. Six weeks later her eyes were examined again, although there had been no additional headaches during this time. Drops were then put in her eyes and for three days she was totally blind. When the vision returned, it was worse than before; she could no longer see to read and could make her way around the house only by the sense of touch. About this time she began to have spells of drowsiness which lasted from one to three days; these were usually preceded by severe frontal headaches with nausea and sometimes vomiting. This train of symptoms appeared every two or three weeks; constipation became more and more severe during this time. After 32 chiropractic treatments she felt somewhat improved, but similar attacks quickly reappeared almost daily for a period of two months. They then cleared up entirely and she was quite well for a time. She grew quite rapidly, put on weight, and her sight improved so that she could recognize pictures on the wall.

About six weeks ago she noticed the right side of her face was numb and for a period of about fifteen minutes her tongue felt twisted and her speech was an unrecognizable jargon. The numbness of the face lasted twenty-four hours. A week later she felt very feverish and was delirious for three or four hours. The doctor took her temperature and found it to be subnormal. The headaches returned at this time and have been both constant and severe up to the time of her admission to the hospital. The headache is sharply localized to the forehead, and more over the right eye and the base of the nose.

Two weeks ago she had a feeling as if something were dropping down in her right forehead.

Physical examination. Patient is rather undersized for her age—she weighs only 82 pounds and is quite poorly nourished. She is so drowsy that it is entirely impossible to attract her attention sufficiently to answer questions.

Neurological examination. The head is larger than normal and suggests a degree of hydrocephalus. There is a definite bulging on the right side of the head. Over this swelling firm pressure with the finger produces a sensation of definite yielding as though the skull were very thin. She is entirely blind in the *left* eye; with the *right* eye she counts fingers with some difficulty. Although there is but little vision remaining in the right eye, there is a suggestive but not definite hemianopsia to the left. The left pupil is fixed and does not react to light; the right pupil reacts slightly to light. When the light is thrown into the right eye there is a slight contraction of both pupils; when light is thrown into the left eye there is no contraction of either pupil. There is some haziness of the discs which are extremely pale and show marked optic atrophy. The veins are full and slightly tortuous; there are no hemorrhages. There is marked spasticity of both sides of the body, but it is much more marked in the left arm and leg. There is a left sided Babinski and a well sustained ankle clonus on the left. No sensory impairment could be elicited. The abdominal reflexes are absent.

X-ray of the head showed generalized convolutional atrophy of the skull. There were no areas of calcification.

Operation, April 5, 1927. Ventricular estimation. The patient was prepared for ventriculography. When an attempt was made to reach the right ventricle into the occipital region 40 cc. of heavy yellow fluid escaped under great pressure, and more could have been obtained if desired. The left ventricle was then tapped. It was very large; the fluid was clear and was still under increased pressure. The diagnosis of a tumor on the right side was, therefore, made from the cystic fluid.

Immediately thereafter the patient was operated upon under local anesthesia. A bone flap was turned down in the right parietal-occipital region. The bone was of paper thinness in the temporal region; in fact there were areas where the bone was entirely missing. This finding explains the feeling of crepitation and yielding to the palpating finger. When

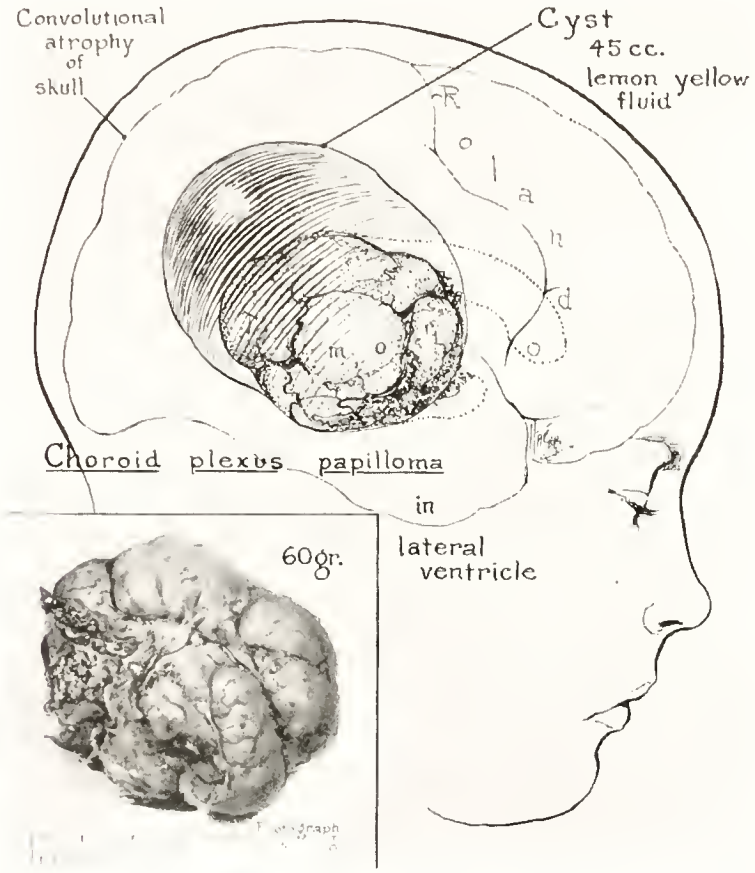

Fig. 47. Operative sketch showing position and size of tumor in Case X

the dura was opened the brain still bulged despite the removal of both ventricular and cystic fluids. In the post-Rolandic region the brain was very pale and soft; the convolutions were greatly flattened and the sulci obliterated. The needle introduced into this region struck the same yellow fluid at a depth of about one-half centimeter. It should be noted that

the Rolandic vein was pushed far forward and was markedly curved owing to the pressure of the tumor situated behind it. The vessels on the softened surface of the brain were doubly ligated with silk and an incision made through the cortex. After aspiration of perhaps one ounce of cystic fluid, one could see a knuckle of solid tumor bulging into the cyst; the tumor was located deeply and anteriorly in the cyst (Fig. 47). It was covered by two large veins which were clipped as they coursed

FIG. 48. Photograph of tumor in Case X after extirpation

from the surface of the tumor to the wall of the cyst. A very hard encapsulated tumor mass was then shelled out with the finger (Fig. 48). There was a rather brisk hemorrhage, which was quickly controlled by packing. The solid portion of the tumor weighed 60 grams. It extended into the temporal lobe and anteriorly along the body of the ventricle; but the tumor was entirely outside the ventricle, which was not opened at any point. It was felt that the tumor probably extended far enough forward to produce direct pressure upon the optic

nerves. Its outskirts were so far in the depth that one could barely reach the anteriormost portion with the finger. The tumor looked very much like a dural endothelioma; it was well encapsulated, very hard, but at no point did it reach the dura.

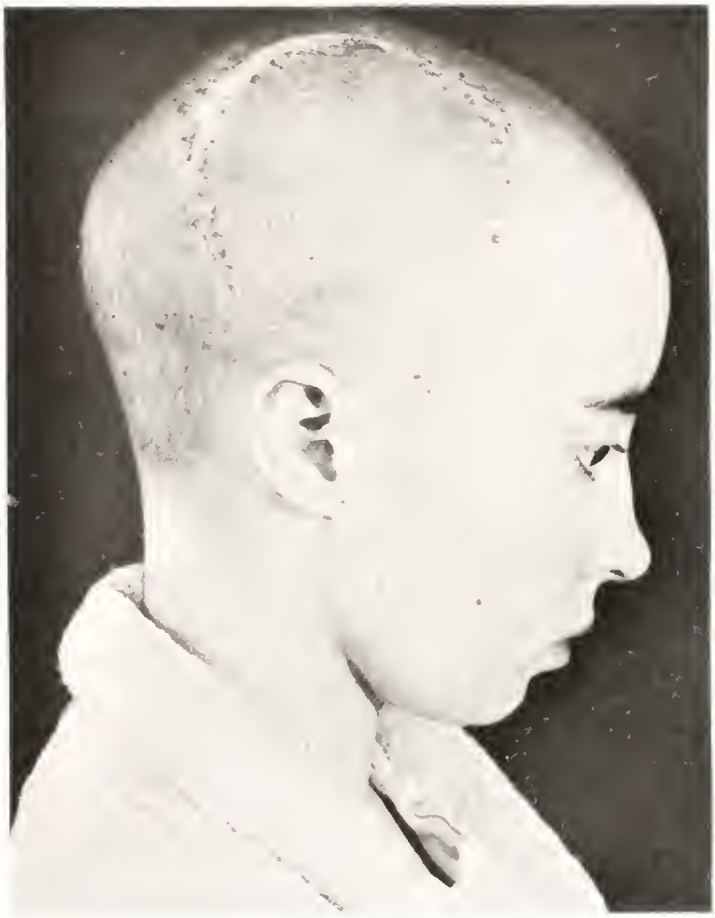

FIG. 49. Photograph showing operative scar in patient from whom tumor of the choroid plexus was removed (Case X).

The bleeding from the tumor's bed was perfectly controlled as long as the pack was left in place, but when it was removed there was oozing from so many points that silver clips were useless. It, therefore, was necessary to leave the pack in place for twenty-four hours. On the following day when the

FIG. 50. Photograph of patient taken five and one-half years after extirpation of tumor (Case X).

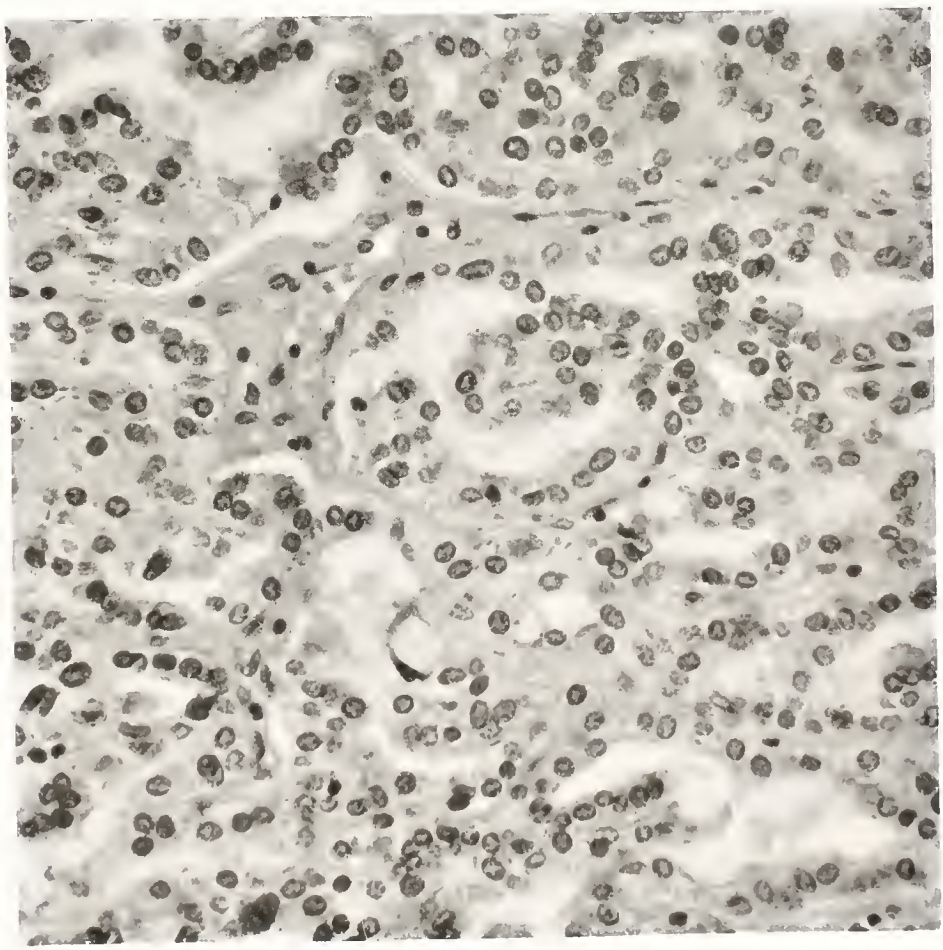

FIG. 51. Photomicrograph of tumor of the choroid plexus (Case X)

pack was removed a large vein was opened, but was easily controlled with a silver clip.

One had a splendid view of the wound and could see the lateral ventricle bulging into the cerebral defect; it, therefore, had not been opened.

The dura was closed, the bone replaced, the galea and skin closed with interrupted sutures of silk.

The patient's recovery from the operation (Fig. 49) was uneventful. She left the hospital seventeen days after the operation and has since remained perfectly well (Fig. 50). Her vision, however, has not returned. She can still read large print. She attended a blind school from which she has now graduated. Full use of the left side of her body was soon restored and has since remained entirely normal. There have been no convulsions.

Microscopic description of the tumor. Choroid plexus epithelium with, for the most part, quite regular alveolar formation (Fig. 51). There are also many solid areas of cells without lumina or alveolar formation. Some areas of colloid are visible. No glial fibers are seen.

Diagnosis. Adenoma of the choroid plexus from misplaced rest outside the ventricle.

Case XI

F. L. Age 12. Admitted: August 5, 1930. Total enucleation of tumor. Discharged: September 3, 1930.

Referred by Dr. Walter M. Otey, of Roanoke, Virginia.

Complaints. Headaches and failing vision.

Family history and past history. Negative.

Present illness. The patient dates her trouble to about a year ago when dull headaches began; they would last from three to twelve hours and were localized to the vertex. At times they spread over the frontal region and were more frequent at night. They seemed to be worse when the patient was quiet and lying down, and to be benefited by sitting or walking. At first the headaches occurred once or twice a week, but they have become

more frequent and have also been more severe. About a year ago double vision developed; this lasted only for a short time and has never reappeared. There has been blurring of the vision, more in the right eye than the left. Attempts to read quickly tire the eyes. She says the pupils are often greatly dilated, and a few moments later they will be quite small. For the past six months there has been dizziness when she

FIG. 52.1. Ventriculogram of Case XI. Anteroposterior view showing dislocation of the ventricular system to the right.

suddenly sits up in bed, or quickly changes her position, or when on her feet. During these spells everything would turn black so that she could not see. For the past three weeks dizziness has been practically constant and there have been many of the blind spells. There have been perhaps half a dozen attacks of vomiting associated with the severe headaches; nausea frequently precedes the vomiting. There have been no convulsions, no aphasia, no tinnitus, no mental disturbances.

Physical and neurological examination. Patient is a very bright girl of 12 years; fairly well nourished and seemingly in good physical condition. There is a moderate degree of papilloedema in both grounds. The margins of the discs are obliterated. The veins are full and tortuous. The change is essentially the same in both discs. Aside from these findings

FIG. 52*B*. Ventriculogram of Case XI. Sharp line of termination of left ventricle at anterior border of the tumor, the position of which is indicated by the dotted circle.

the neurological examination is entirely negative. The reflexes are normal.

X-ray of the head is negative.

Ventriculography. August 15, 1930 an air injection was performed. The ventricular system was small. Air was injected into the right ventricle which was of normal size and shape. The ventricular system was dislocated towards the

right, indicating a left cerebral tumor (Fig. 52*A*). The body of the left lateral ventricle was abruptly closed just back of the foramen of Monro; the air would not pass this line, which indicated the anterior limit of the tumor (Fig. 52*B*).

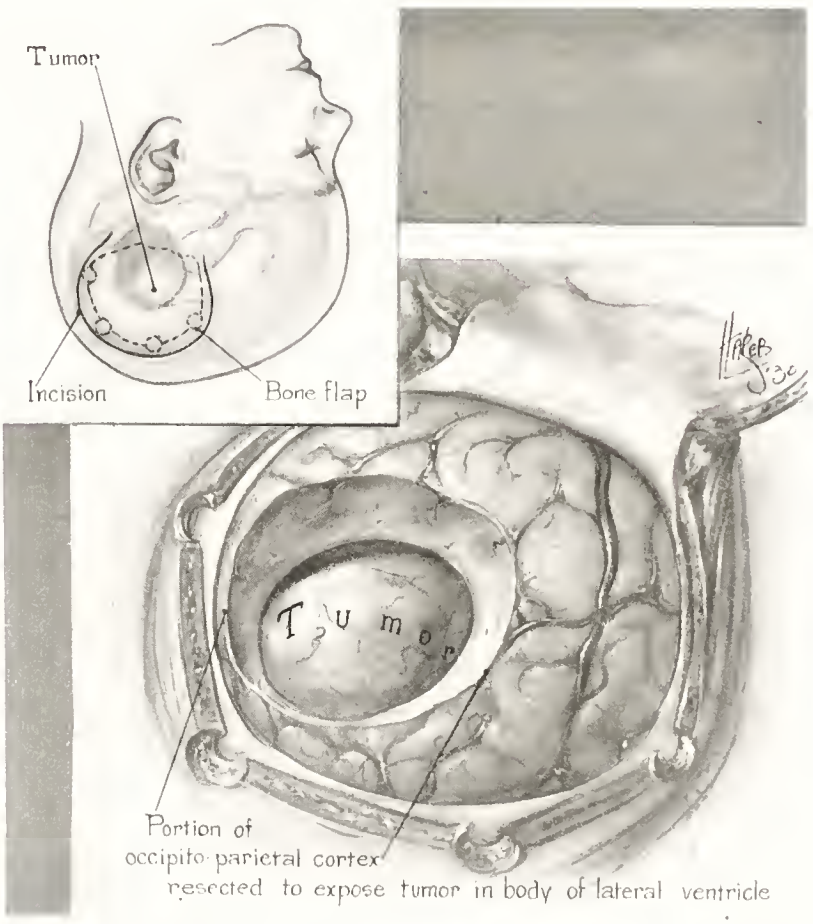

FIG. 53. Drawing showing circular cortical defect uncovering the tumor in the post-Rolandic area (Case XI). Inset shows the position of the craniotomy approach and the location of the tumor.

Operation, August 15, 1930. As the head was being cleaned for operation we remarked about the excessive distance between the ear and the midsagittal line on the left side as compared to the right: such a deformity must be due to a congenital tumor which had caused the skull to be misshapen in early life.

Under avertin anesthesia a large bone flap was turned down

in the left parieto-occipital region (Fig. 53). The bone was exceedingly vascular; it cozed freely from numerous points over the parietal eminence. This great vascularity suggested that a tumor of long standing was immediately beneath.

In the occipital region the cortex was paler than elsewhere, but there was no sign of a tumor. A ventricular needle was

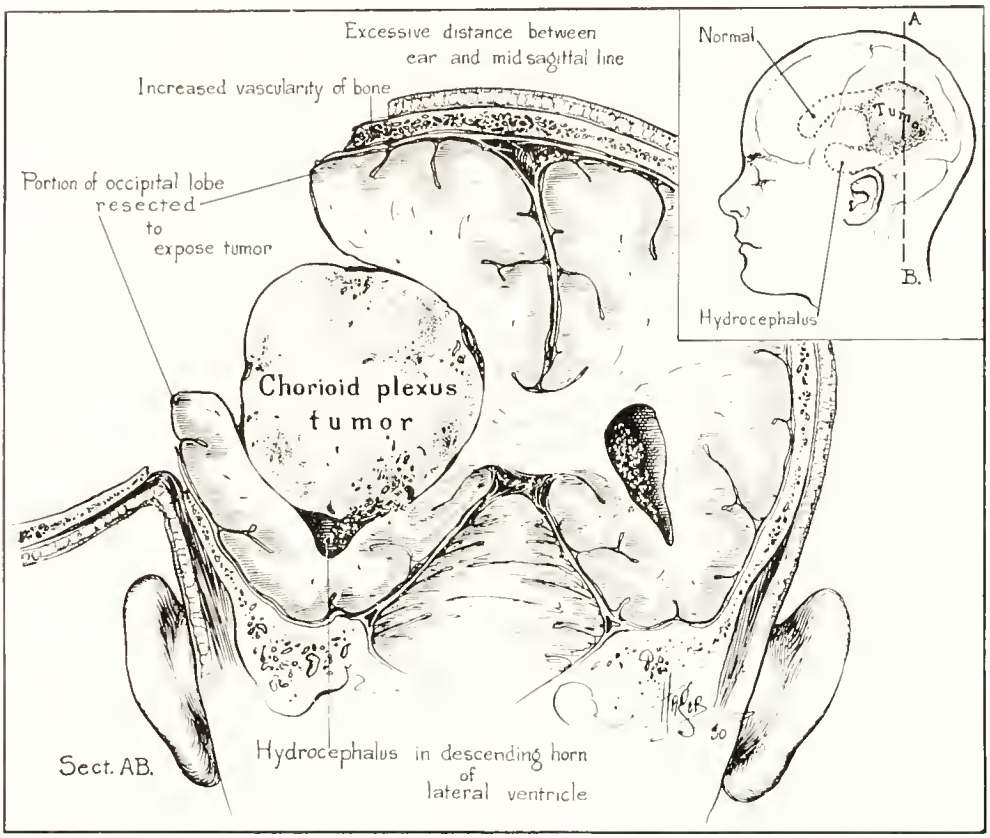

Fig. 54. Sketch of anteroposterior view to indicate the position of the tumor (Case XI) and the cortical defect through which it was exposed.

inserted and encountered a very firm resistance at a depth of 3 cm. The tumor was so hard that the needle could not be passed through its capsule without using undue force. A large circular area of cortex was excised from the posterior part of the parietal and the anterior part of the occipital lobes (Fig. 53). The cortical defect must have included the supramarginal and angular gyri. The cortical defect measured about

5 x 5 cm. The surface of the tumor was then in view (Fig. 54). It was perfectly encapsulated, smooth, and of a grayish-brown color. The finger was passed around the tumor and it was quickly extirpated from its bed. The tumor extended into the temporal lobe, forward under the Rolandic area and backward almost to the tip of the occipital lobe; mesially it reached the falx. The ventricle was now wide open. There was a brisk hemorrhage, the source of which was soon seen to be the choroid plexus to which the tumor had been attached; it was in fact, the sole attachment of the tumor. With tampons of moist cotton the bleeding was suppressed and finally the bleeding vessels were picked up with forceps and clipped. Both veins and arteries in the choroid plexus were large and tortuous. We could now see the remains of the choroid plexus in the descending horn of the ventricle and its remains in the body of ventricle with no choroid plexus between. The tumor, therefore, had arisen from the glomus of the choroid plexus and this was later identified in the wall of the tumor. The descending horn of the ventricle was markedly dilated because the tumor had obstructed the outflow of cerebrospinal fluid. Several oozing points along the cut surface of the brain were controlled with the electro-cautery.

The wound was entirely dry when closure was begun. The dura was closed tightly and the bone flap replaced; no drainage.

Gross description of tumor. The tumor was white, exceedingly hard, incompressible, and almost perfectly round (Fig. 55). It was the largest of the series of ventricular tumors, weighing 124 grams. The surface was everywhere perfectly smooth except where the choroidal vessels of the body and descending horns entered the tumor separately and about 3 cm. apart. Here was a roughly oval area, slightly more pink and somewhat trabeculated. Its appearance suggested that the strands of choroid plexus, which surrounded the entering veins at these two widely separated points, would probably also be found in the underlying tumor tissue, but this suggestion was not borne out by the microscopic findings. There

could be no question of the origin of the growth from this structure.

On cross section the tumor was firm, hard and, except for a single small cyst with mucous content, was of fairly homogeneous appearance (Fig. 56, *A* and *B*) although there was some necrosis. The capsule of the tumor was 2 or 3 cm. thick and everywhere intact.

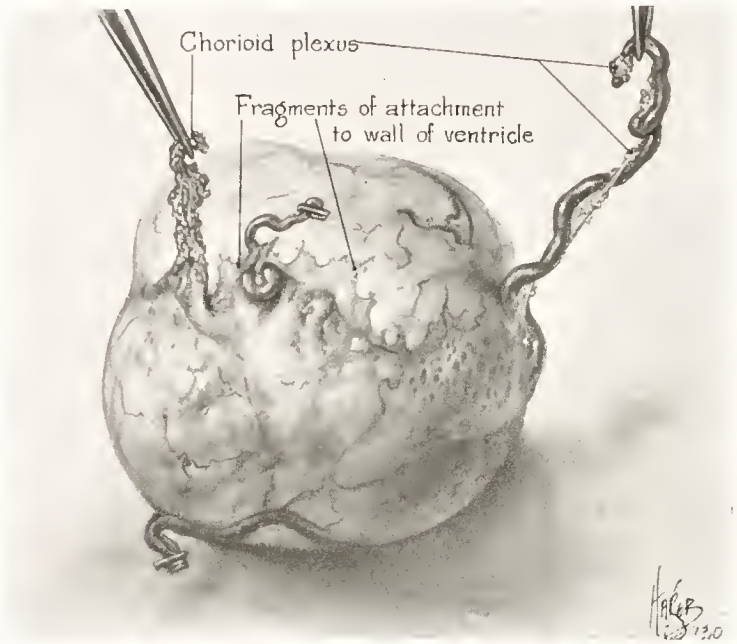

FIG. 55. Drawing of tumor after its removal (Case XI). Note the choroid plexus entering the tumor on each side.

Microscopic report of the tumor. The tumor is made up of compact fibrous tissue (Fig. 57), frequently arranged in whorls. Many small cell nests, doubtless of connective tissue type, are present throughout the sections. The cells stain deeply. There are few calcareous bodies. No sign of choroid plexus or ependymal epithelium can be seen. A thin bed of fibrous tissue lines the tumor, but externally there is no ependymal lining. Areas of colloid are present throughout.

Post-operative course. Patient made an uneventful recovery (Fig. 58) except for the defects incident to the removal of the

FIG. 56. *A* and *B*. Photograph of surface and cut section of case (Case XI)

large circular area of cortex in such an important area of the brain. After the operation she had total anomia, total alexia, total agraphia, total apraxia, and homonymous hemianopsia which was complete for colors but not for form. Some vision

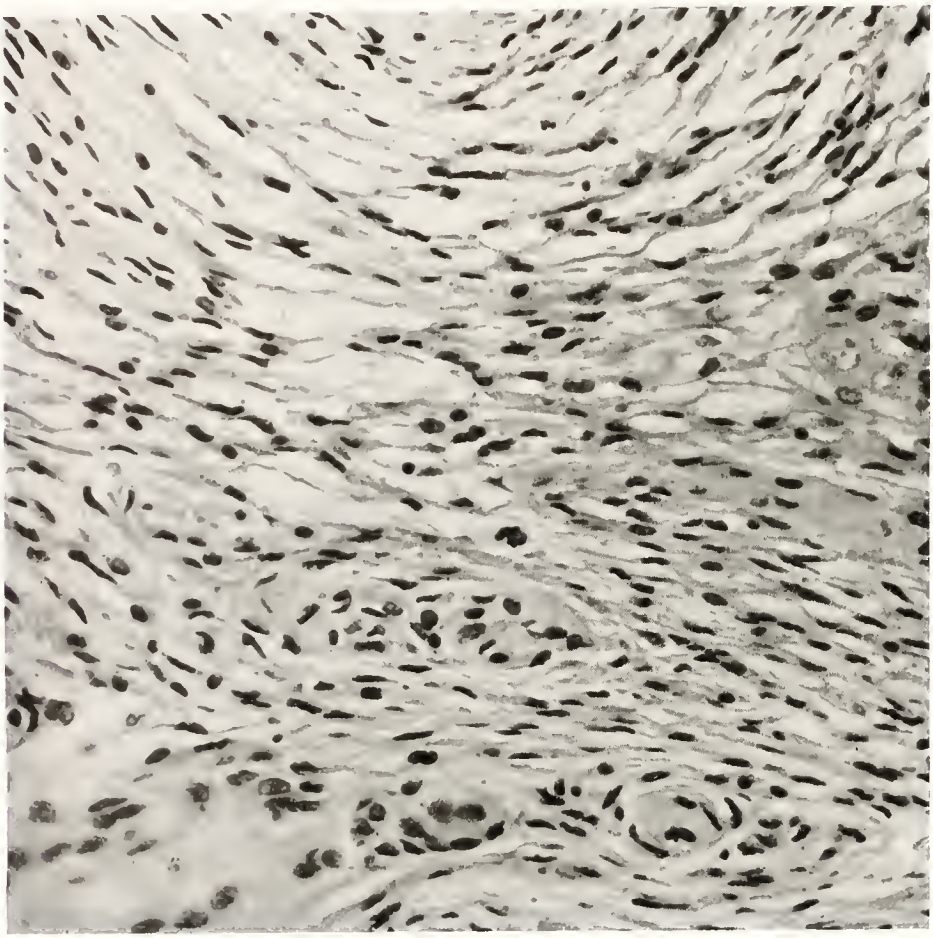

FIG. 57. Photomicrograph of tumor Case XI

for form remained in the temporal half of the right eye (see chart).

Nine months after the operation patient's apraxia had cleared, anomia had improved tremendously; she was able to read a great deal, though this has been the function slowest to return. At no time has there been auditory aphasia, and her motor aphasia has been restricted to nouns. Her mind is

exceedingly clear and she carries on a conversation without hesitancy, except for nouns. The proper nouns give her more trouble than the common nouns, most of which have now returned. The disturbance of speech will not be detailed here,

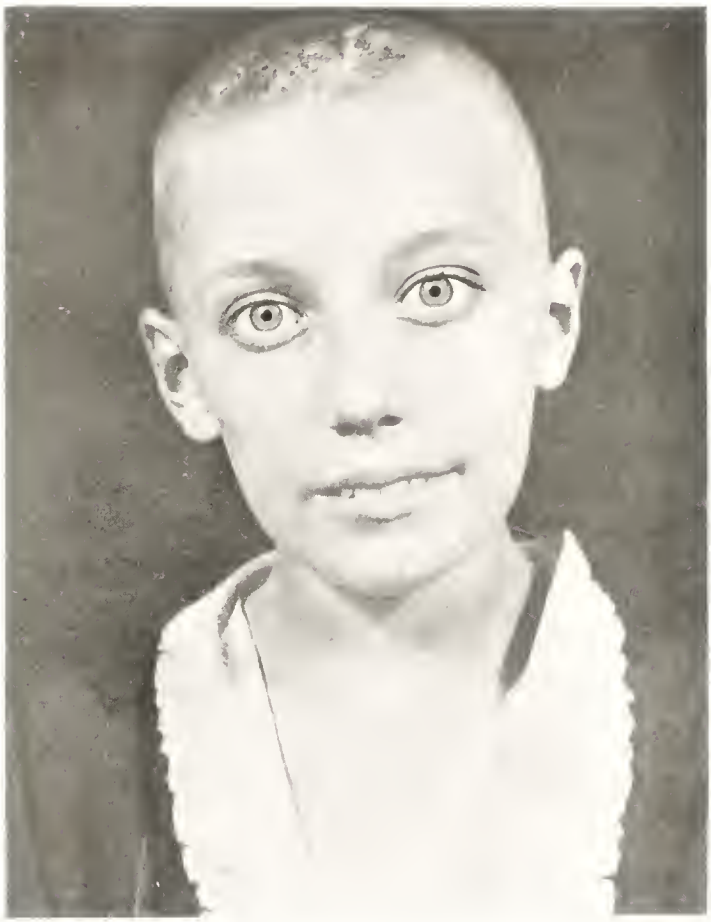

FIG. 58. Photograph of patient taken at the time of discharge from the hospital

but will be considered in a subsequent communication dealing with aphasia resulting from operative defects.

Diagnosis. Pure fibroma of choroid plexus.

Case XII

L. A. Age 35; admitted May 10, 1933. Total enucleation of tumor. Discharged May 29, 1933. Referred from the

United States Naval Hospital, Washington, D. C., with the diagnosis of an intracranial tumor.

Complaints. Headache, loss of hearing, and loss of vision.

Family and past histories. Negative. The patient is an aviator and ascribes his loss of hearing, which is bilateral, to the noise of the engine.

Present illness. Five months ago patient noticed that he was a little clumsy with his *right* leg. This was evident by overstepping objects, or lunging when he came in contact with an irregularity in the ground. There was no actual weakness of the leg and no staggering, but this condition has remained practically unchanged to date. There is no associated loss of sensation. About this same time severe bifrontal headaches began, perhaps more on the right; they were dull, penetrating, and more or less constant. Although the headaches have been generalized, they have perhaps localized more to the region of the right mastoid, and a local tenderness over the mastoid has been noted at times. The headaches began in the early afternoon and grew worse toward evening; they now occur almost daily; they are relieved by aspirin; they are not accompanied by vomiting or nausea; they are aggravated by straining and coughing. At this time an infection of the right antrum was found and drained. This was repeated on several subsequent occasions. There seemed to be some amelioration of the headache following these treatments. About a month ago he noticed diminution of vision. He was apt to miss several words in a sentence; usually the words were at the beginning of the sentence. A diminution in the central vision was also noticed; this was not improved by glasses. More recently there has been blurring and drawing of the eyes when reading. About a month ago he had low pitched tinnitus in the left ear for three nights; he thinks this occurred with the greatest intensity of the headache. This disappeared and recurred again for a similar period. Recently it has been absent. About two weeks ago he had a queer sensation of a bitter taste in the back of his mouth; it was more or less constant but inten-

sified when food was taken. He has noticed that this queer taste destroyed the normal flavor of foods and no food has since tasted normal to him. He describes the taste as that of floor-wax.

Physical and neurological examinations. Patient is a large, robust young man in perfect physical condition. At the United States Naval Hospital the following findings were obtained: Papilloedema of high grade; an abrupt loss of high tones in both ears, more pronounced in the right. At one time some loss of sensation was observed in the right trigeminal domain and possibly a loss of function in the muscles of the right side of the face. It was their impression that he had a right cerebellopontine tumor.

However, at the time I saw him no loss of function could be discovered in either the fifth or seventh nerve, and there were no cerebellar signs whatever. On the other hand, a very definite sharp-cut right homonymous hemianopsia for color was disclosed. This, however, was the only positive finding of any type, except papilloedema of both eyes, and essentially the same in each eye. On more careful examination by Dr. MacLean the same right homonymous hemianopsia for color was found (Fig. 59). His visual acuity was 20/50 in the right eye and 20/70 in the left eye. The audiometer curve here was essentially the same as that taken at the U. S. Naval Hospital (Fig. 60). The vestibular response was essentially the same on both sides.

There was some loss of vibratory sense in the left ankle and knee, but no other sensory finding. This change was not regarded as important at the time, but it was probably significant. However, his complaint of clumsiness of the *right* leg would have referred the lesion to the other side. There were no other positive neurological findings. His blood pressure was slightly elevated, being consistently around 150. Wassermann reaction from the blood was negative.

Impressions. Brain tumor (1) located in the *left* temporal lobe because of the very definite right homonymous hemi-

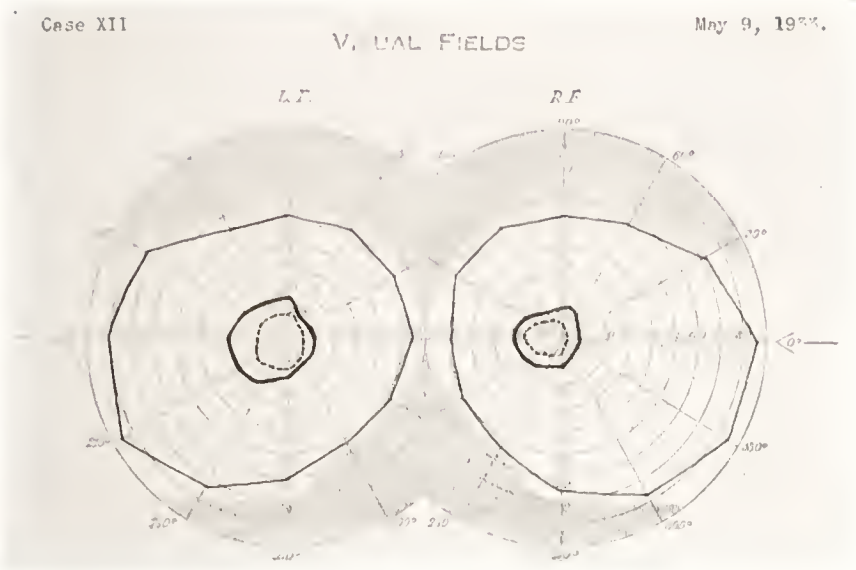

FIG. 59. Visual fields (Case XII) showing right homonymous hemianopsia for color. It was erroneously assumed that this finding indicated a tumor in the left cerebral hemisphere: the tumor was actually in the right.

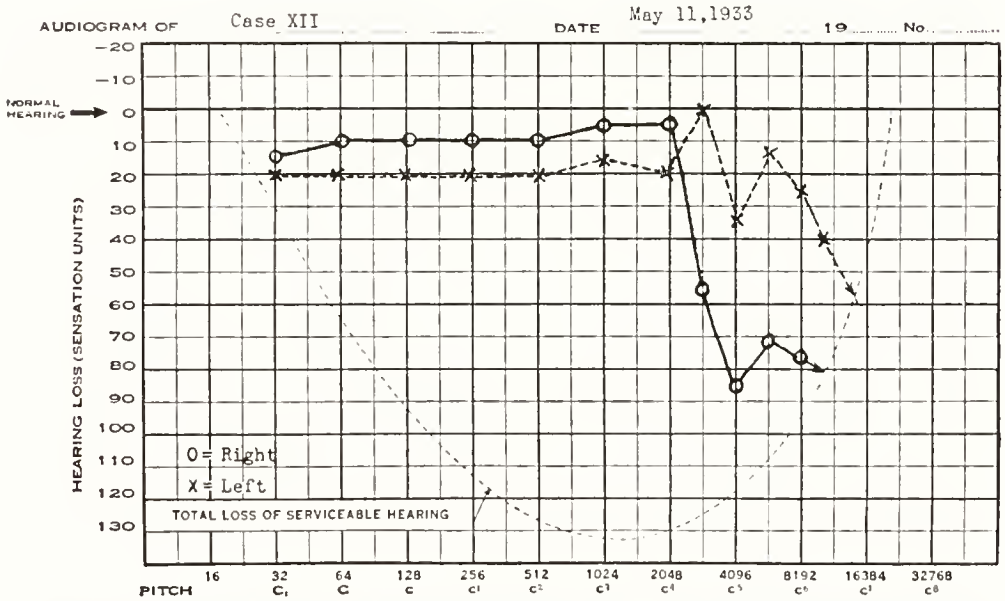

FIG. 60. Audiometer curve showing the abrupt loss of high tones in both ears, but more on the right. This finding is thought to indicate a lesion in the region of the geniculate bodies.

anopsia for color. It was thought the subjective clumsiness of the right leg might correlate with this finding. However, the evidence was not sufficient to warrant a left craniotomy. (2) The audiometer curves suggested a tumor of the pineal or third ventricle. Could the right homonymous hemianopsia be due to the effect upon the geniculate bodies? More precise localization by ventriculography was indicated.

Ventriculography was performed May 12, 1933. The fluid spurted under enormous pressure from the left ventricle, which was also much enlarged; 75 cc. of fluid were aspirated and an equal amount of air injected. The ventriculograms showed the following: the left ventricle was dilated throughout; only a very small amount of air reached the anterior horn of the right lateral ventricle; its size was greatly reduced and its shape though normal was in striking contrast with the big rounded ventricle on the left. The third ventricle was filled only in the anterior half. There was a large round sharply-defined filling defect in its posterior half. The entire ventricular system was pushed to the left side and the third ventricle was oblique.

Ventriculographic diagnosis. Tumor in the posterior part of the *right* cerebral hemisphere (Fig. 61*B*); it was deeply located producing a filling defect in the third ventricle (Fig. 61*C*); this obstruction in turn was causing dilatation of the left lateral ventricle; the size of the right lateral ventricle was greatly reduced by the volume of the tumor on this side (Fig. 61*A*). It will be noted that the ventriculograms localized the lesion to the *right* side and denied any localizing value to the right homonymous hemianopsia for color, which could hardly be questioned because it was found by two observers independently. We had always regarded hemianopsia as a sign of almost pathognomonic significance.

Operation, May 12, 1933. A bone flap was turned down in the right parietal-occipital region. The dura was under excessive pressure. This was in large part relieved by tapping the left lateral ventricle through an opening placed over the

anterior horn. When the dura was reflected there was no sign of a tumor. There was perhaps a little pallor in the post-Rolandic area. A ventricular needle was passed through the cortex and at a depth of 5 cm. a resistance was encountered. A nasal dilator was passed through the tract of the needle,

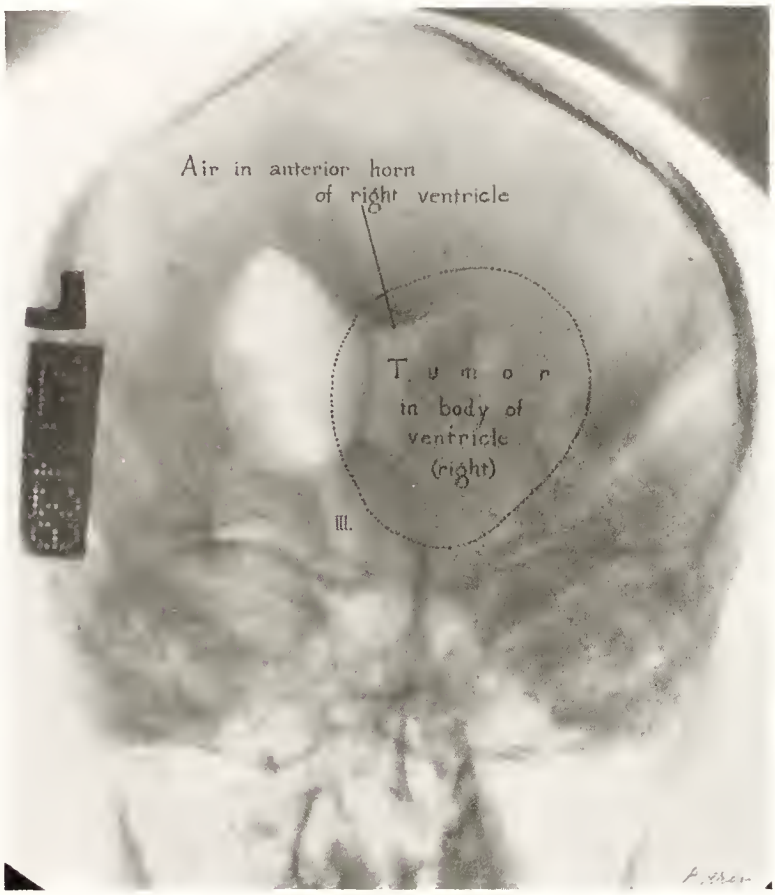

FIG. 61.1. Ventriculogram of Case XII. Anteroposterior view. Note the huge left ventricle, the small remains of the right ventricle and the oblique third ventricle. There is a slight dislocation of the ventricular system to the left side. This is best indicated by the oblique third ventricle.

and at the same depth the posterior border of the tumor was visible. The finger was then passed through this defect. The tumor was very hard, seemingly perfectly encapsulated; it was possible to barely reach the under-surface of the tumor. A circular defect, probably 4 cm. in diameter, was made just

mesial to the Sylvian vein and slightly posterior to the post-Rolandic area; it was certainly well back of the motor center. It was made as far posteriorly as possible in order to avoid injury to the motor tracts; this made the approach to the tumor from behind, rather than directly above it. With this exposure the

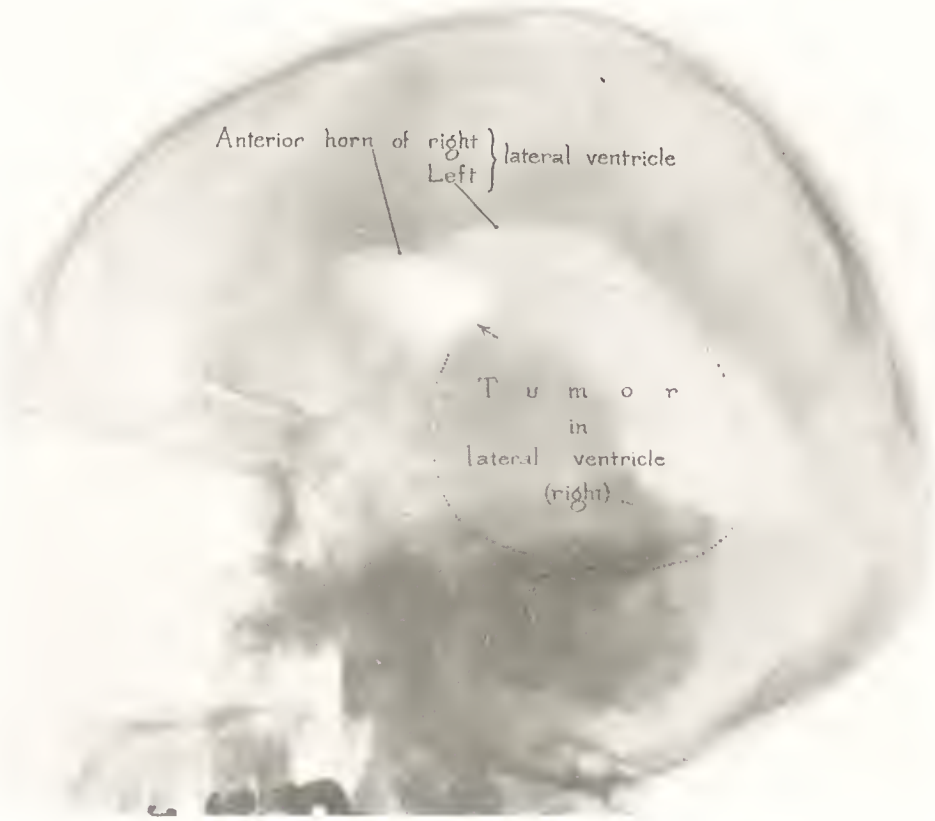

FIG. 61*B*. Ventriculogram of Case XII. Lateral view. Note the abrupt termination of the shadow of the left ventricle, well anteriorly. This is the anterior border of the tumor. The region of the tumor is indicated by the circular dotted lines.

finger was again inserted and could be passed around the tumor for some distance on all sides without provoking any bleeding. It was clearly a well encapsulated tumor, and although its position suggested that it was in the lateral ventricle, the walls of the lateral ventricle could not, as yet, be definitely seen. The tumor was so hard that it was impossible to extirpate its interior with the curette. The cerebral defect was

ample to permit the introduction of two fingers, one on the inferior, the other on the superior surface of the tumor. Two fingers were used in order to compress the tumor and draw it posteriorly; thus leverage on the brain was reduced to a minimum (Fig. 62). Extirpation with a single finger would have

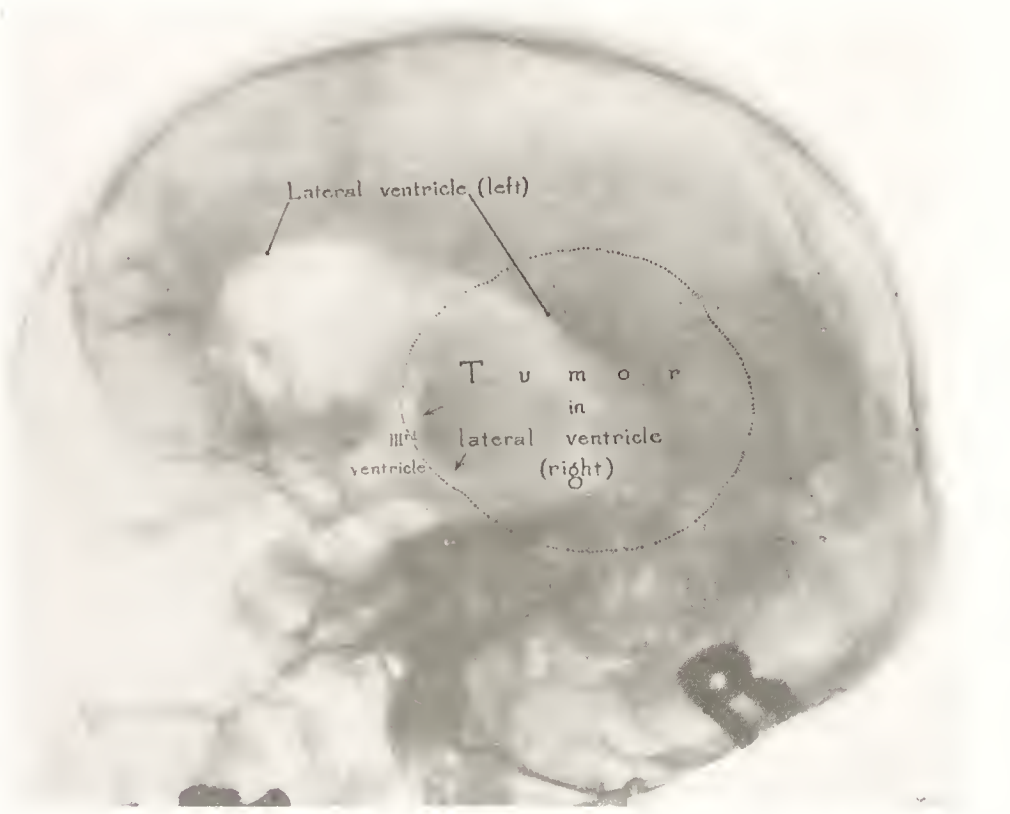

FIG. 61C. Ventriculogram of Case XII. Lateral ventriculogram showing the filling defect in the posterior half of the third ventricle. It was this occlusion that produced the large left lateral ventricle.

necessitated application of leverage and perhaps would have injured the overlying motor tracts. The tumor was so slightly adherent to the surrounding tissue that it was extirpated without difficulty, but it was followed by a furious hemorrhage. This was quickly controlled with cotton packs, after which the cotton packs were gradually removed. The bed from which the tumor was removed was aspirated and three or four large

bleeding veins could be seen dangling in the cavity. These were picked up with forceps and thrombosed with the electro-

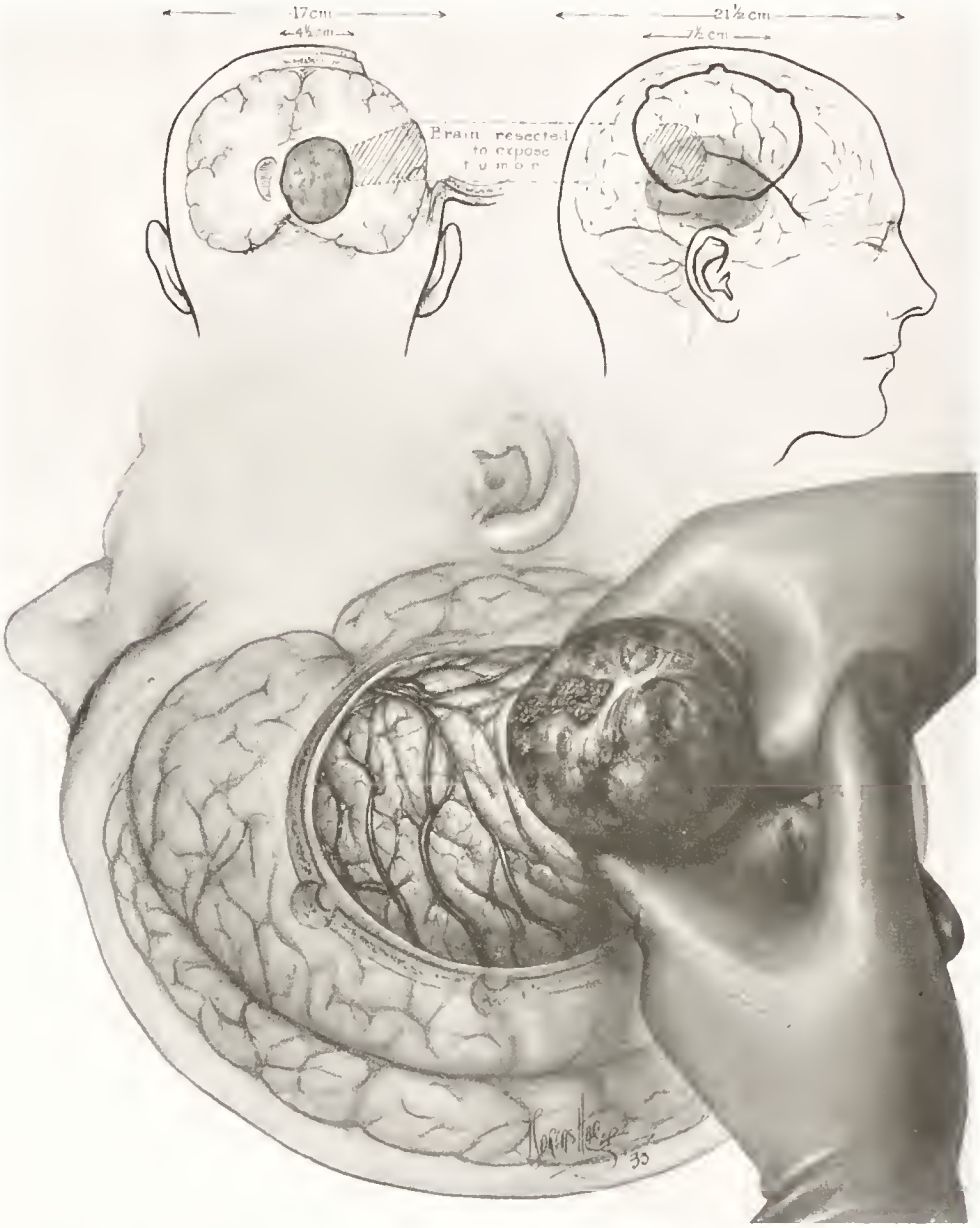

FIG. 62. A drawing of the operation in Case XII, showing method of enucleation of the tumor by the index finger of each hand.

cautery. The wound was eventually perfectly dry. One could then see that the tumor was within the vestibule and body of

the lateral ventricle and the choroid plexus in the body of the ventricle was seen dangling in the cavity (Fig. 63). This structure was clipped (Fig. 64) and thrombosed at its point

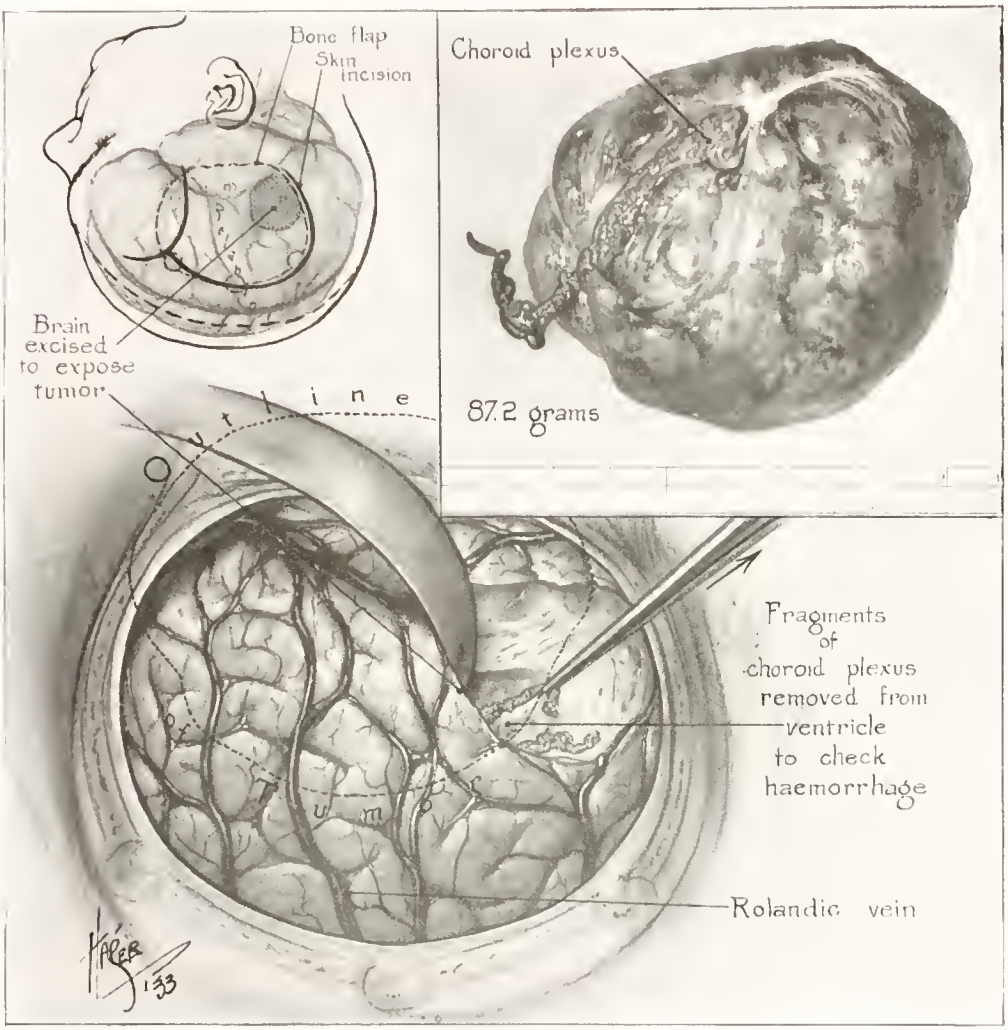

FIG. 63. Drawing to show site and relative size of the cerebral defect through which the tumor in Case XII was exposed and removed. The inset shows the drawing of the tumor with choroid plexus, from which it arose, attached.

of attachment to the wall of the ventricle and the excess excised. The patient's blood pressure was very low; he was given an intravenous injection of glucose and a transfusion during the operation. The cerebral defect was filled with

Ringer's solution, the dura closed tightly, the bone flap replaced. The wound was closed without drainage.

The patient's condition the following morning was good. He was able to use the left leg as well as the right. There was slight weakness of the left arm and hand, though both could

FIG. 64. Anteroposterior x-ray showing silver clips placed upon the choroid plexus at the time of operation and indicating the approximate position of the tumor.

be used. Three days after the operation it was necessary to evacuate the necrotic tissue. Following this there were perhaps a dozen localized convulsions, beginning in the left face and arm; two of these became generalized. There after his general condition and motor weakness rapidly improved.

He was discharged from the hospital May 29, 1933, seventeen

days after the operation (Figs. 65 and 66). He had complete right homonymous hemianopsia, which was the only objective sequela of the operation. The preoperative left hemianopsia for color had disappeared. The function in the left arm was

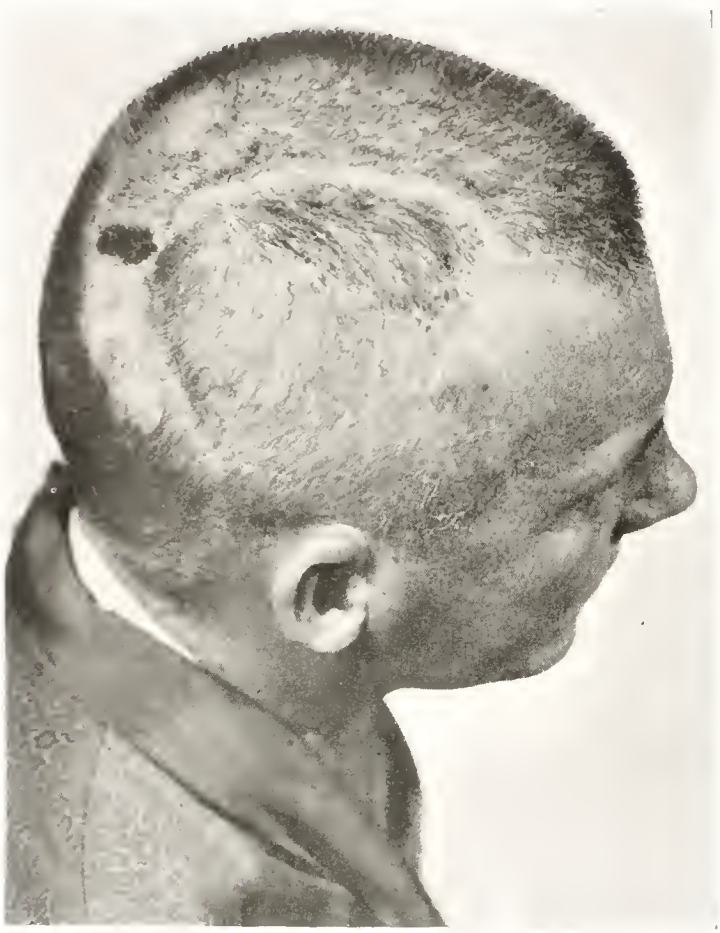

FIG. 65. Photograph of patient (Case XII) showing site of operative approach

now normal. The audiometer curves remained as before operation (Figs. 67 and 67A).

The tumor weighed 95 grams; it was almost perfectly oval, 8 cms. long and 6 cms. wide (Fig. 68). It was firm and fairly elastic. Attached to it was choroid plexus which grew directly into the tumor, exactly as in Cases XI and XIII. The length of the tumor was considerably more than one-third of the total

FIG. 66. Photograph of patient (Case XII) taken at time of his discharge from the hospital.

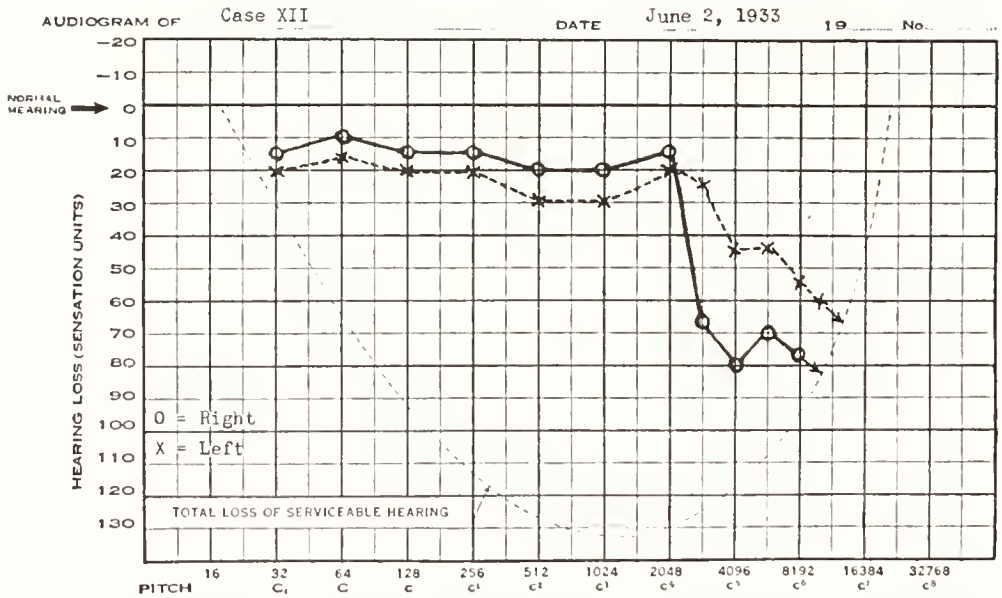

FIG. 67. Audiometer curves (Case XII) at time of discharge from the hospital: they were almost exactly the same as before operation. Six weeks later audiometer curves were sent from the Naval Hospital and at that time they still remained unchanged.

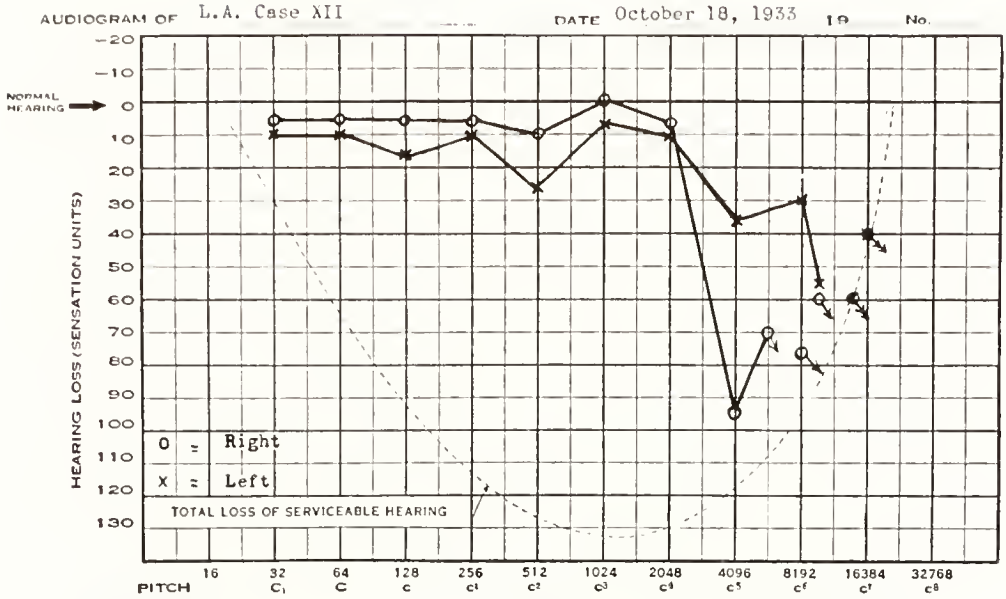

FIG. 67.1. Audiometer report (Case XII) five months after operation

FIG. 68. Photograph of cut section of tumor from Case XII

length of the cranial chamber; which measured (by calipers) 20 cm. from the inion to the nasion.

Microscopic note. Section is made up of fibrous tissue with spindle shaped nuclei throughout (Fig. 69). In many places the tissue is very compact; in others, vacuoles of varying size

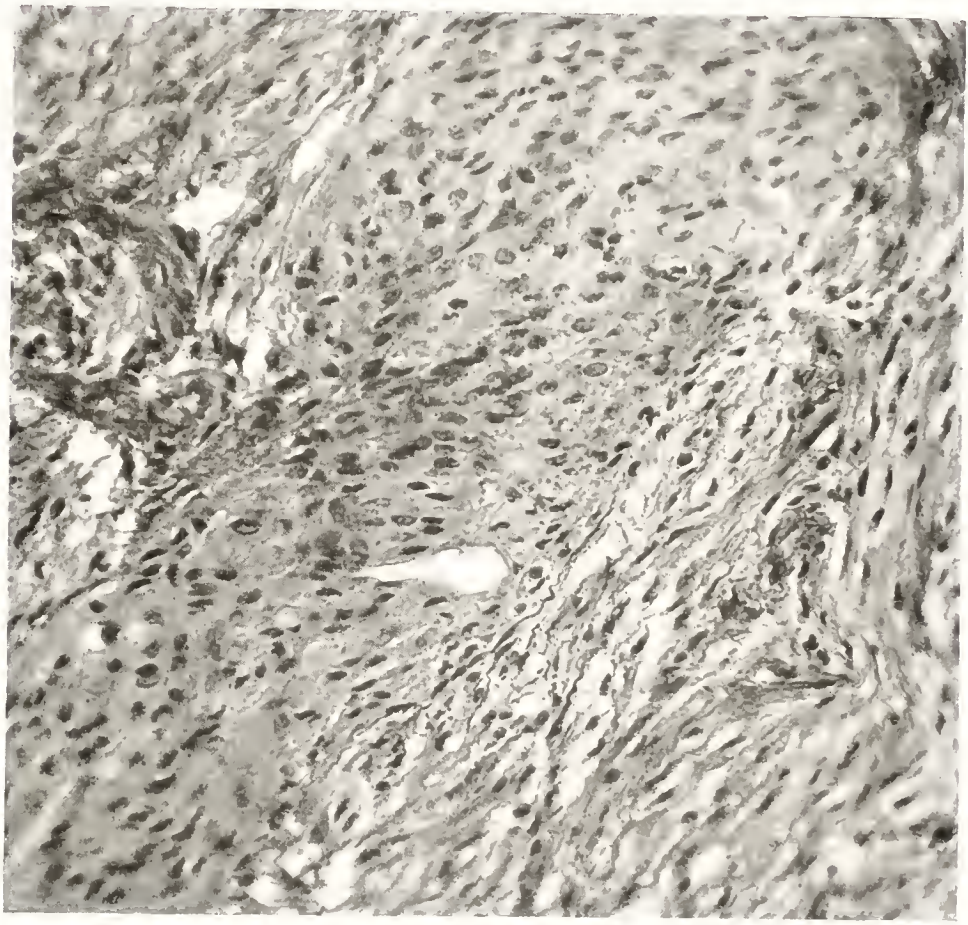

Fig. 69. Photomicrograph of tumor (Case XII)

separate the fibers. Throughout the tumor the fibers are frequently arranged in whorls. A single psammoma body is seen in three large sections. In one section there are occasional epithelial remains suggesting incorporated choroid plexus. No epithelial layer covers the tumor. The microscopic picture is identical with that of Case XI.

Diagnosis. Pure fibroma of choroid plexus.

Case XIII

J. W. F. Age 48. Admitted: September 15, 1933. Total enucleation of tumor Discharged: October 5, 1933.

A large, well-nourished, normal appearing man of 48 years, was referred by Dr. William S. Sims, Jr., of Washington, D. C., with the diagnosis of a brain tumor.

Complaints. Headache, vomiting, unsteadiness of gait, numbness of the left hand and loss of libido.

Family history and past history are negative.

Present illness. Began thirteen months ago at the time of his marriage when, for no apparent reason, libido was lost and has never since returned. One or two weeks later vomiting suddenly appeared without nausea and without headache. This recurred quite regularly about once a week. A few weeks later headaches developed in the frontal region back of both eyes, but was always greater on the right and especially back of the right eye. The headache would last from a few hours to twelve or more, and was frequently, but not always, associated with vomiting. During the next eight months the headaches gradually increased in severity and frequency. He then began to grow listless and to lack his usual vigor. He also slept more than usual. At this time he was seen in the Diagnostic Clinic of the Johns Hopkins Hospital, but nothing was found to explain his headaches or other symptoms. There were no neurological findings at that time.

Five months ago his lethargy markedly increased. He was too tired to go out during the evening and when trying to read he would fall asleep. At the same time he began to have difficulty in making legal decisions (he was a lawyer) and his partners began to comment upon it. He then noticed that he had difficulty in walking in the dark; he also ran into objects (probably due to hemianopsia). He began to stumble, especially when going up and down stairs and had difficulty in maintaining his balance. Clumsiness in the left arm and leg was then noticed. His wife had observed unsteadiness of

gait, but cannot recall whether he deviated to one side more than the other. One and one-half months ago in reaching in his pocket with his left hand he found that coins and other objects could not be recognized; this has since persisted. He would also "lose his hand" and find it in unusual positions. The hand seemed to be apart from the rest of his body.

The patient has not observed any change in his vision, although recently it was pointed out to him that he could not see to the left. Previously his wife had observed that in crossing the street he did not see automobiles approaching, but could not tell that his vision was more affected on one side than the other. His headaches have become more and more severe and occur every few days. There have been no convulsions, no diplopia, no tinnitus, and no disturbance of speech.

Physical examination. The patient is a tall, well developed, well-nourished man; does not look at all ill. Blood pressure is normal; pulse, temperature and respirations are normal.

Neurological examination. There is beginning papilloedema in the right eye as evidenced by blurring and slight fullness and tortuosity of the vessels, the left disc looks quite normal. There is complete homonymous hemianopsia to the left (Fig. 70); a definite weakness (of central type) of the left side of the face. The left arm and leg are perhaps 20 per cent weaker than the right. Light touch is lost over the entire left side of the body, including the face, but stronger touch is well recognized. Pin point is dull and neither heat nor cold is recognized on the entire left side, including the face. Sense of position of the fingers and toes on the left is absent. There is complete astereognosis in the left hand. The deep reflexes are increased on the left. There is no ankle clonus or Babinski, but a positive Oppenheim is present on the left. X-ray of the head is negative. The audiometer test shows loss of high tones in both ears (Fig. 71). Wassermann reaction from the blood is negative.

Diagnosis. Brain tumor right parietal lobe.

Operation, September 16, 1933. Since the localization of the

patient's tumor could be safely determined by the neurological signs, ventriculography was not necessary. Under avertin anesthesia a moderate sized bone flap was turned down in the right parietal-occipital region. When the exceedingly tight dura was opened the brain herniated markedly. The entire exposed cortex was paler and softer than normal and the subarachnoid spaces contained no fluid. Although the tumor was not visible, there could be no doubt that it lay directly beneath.

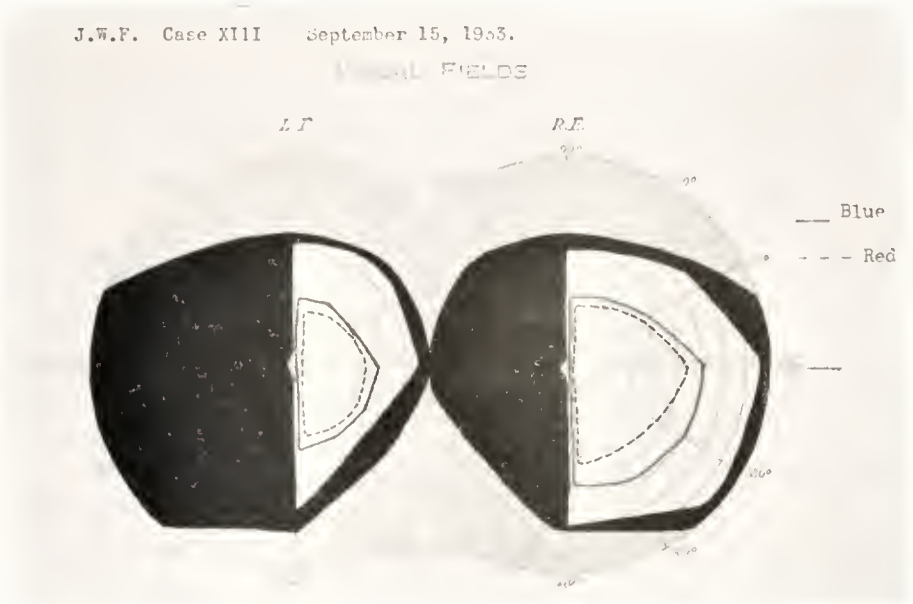

FIG. 70. Complete hemonymous hemianopsia was an outstanding sign in the localization of the tumor in Case XIII.

A needle was inserted through the cortex just mesial to the Sylvian vein and well behind the Rolandic vein. At a depth of 4 cm. a hard tumor was encountered. An oblique transcortical incision was made directly backward and mesially. A grayish-white, hard, nodular and perfectly encapsulated tumor presented when the incision was widened by a nasal dilator; it looked exactly like the two preceding large fibromata of the choroid plexus. The tumor was quickly extirpated with the finger; it had no firm attachments (Fig. 72). A moderate hemorrhage followed the extirpation; after removing the cot-

ton-pack several bleeding veins were picked up with the forceps and sealed with the cautery.

One could now see that the tumor had been entirely within the ventricle. The body, posterior, and descending horns of

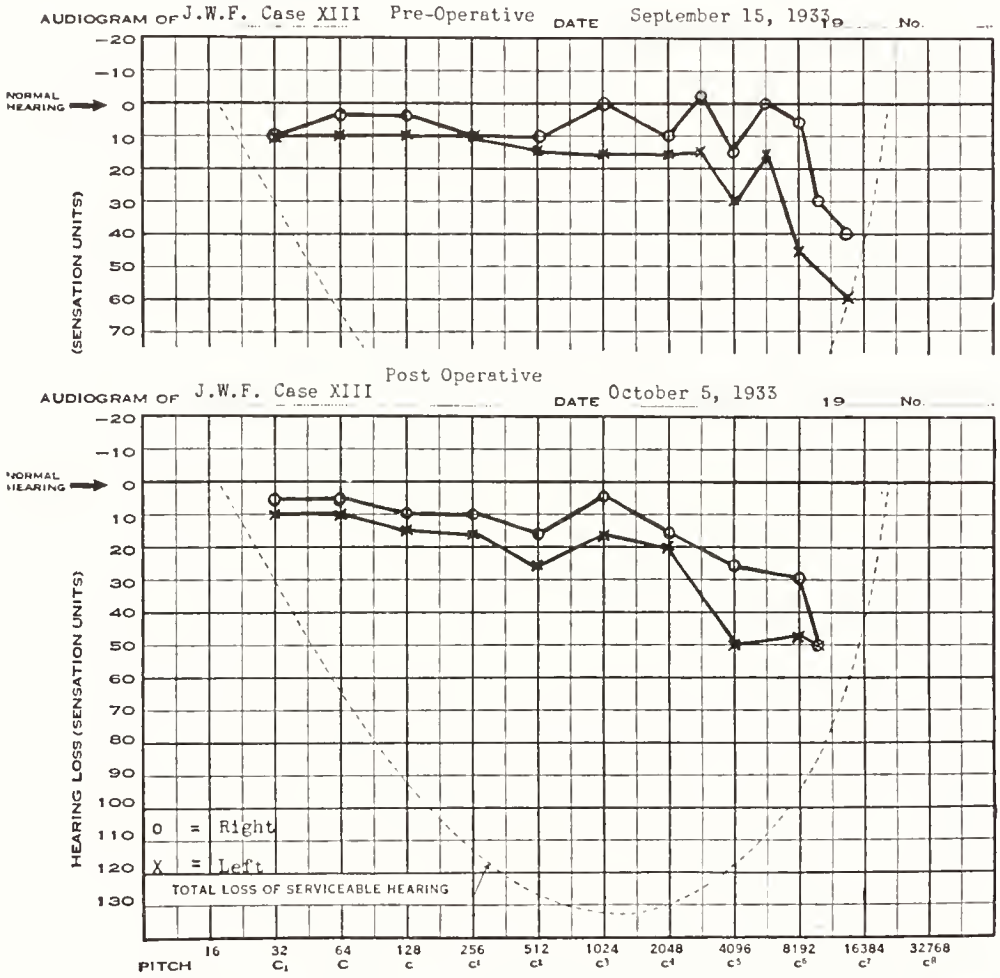

FIG. 71. Audiometer curves from Case XIII before (above) and after (below) operation, showing slight drop in high tones of both ears. Apparently the loss was slightly intensified by the enucleation of the tumor.

the ventricle were in view and everywhere the walls were smooth and glistening. The glomus of the choroid plexus was absent. There was some slight bleeding from the torn ends of the plexus in the body and the descending horn where the tumor had been detached. Since there was ample room in the cranial

chamber the dura was closed and the bone flap replaced. The wound was closed without drainage.

Because of a declining blood pressure patient was given a transfusion of glucose during the operation, and later a transfusion of blood. His condition remained good at all times.

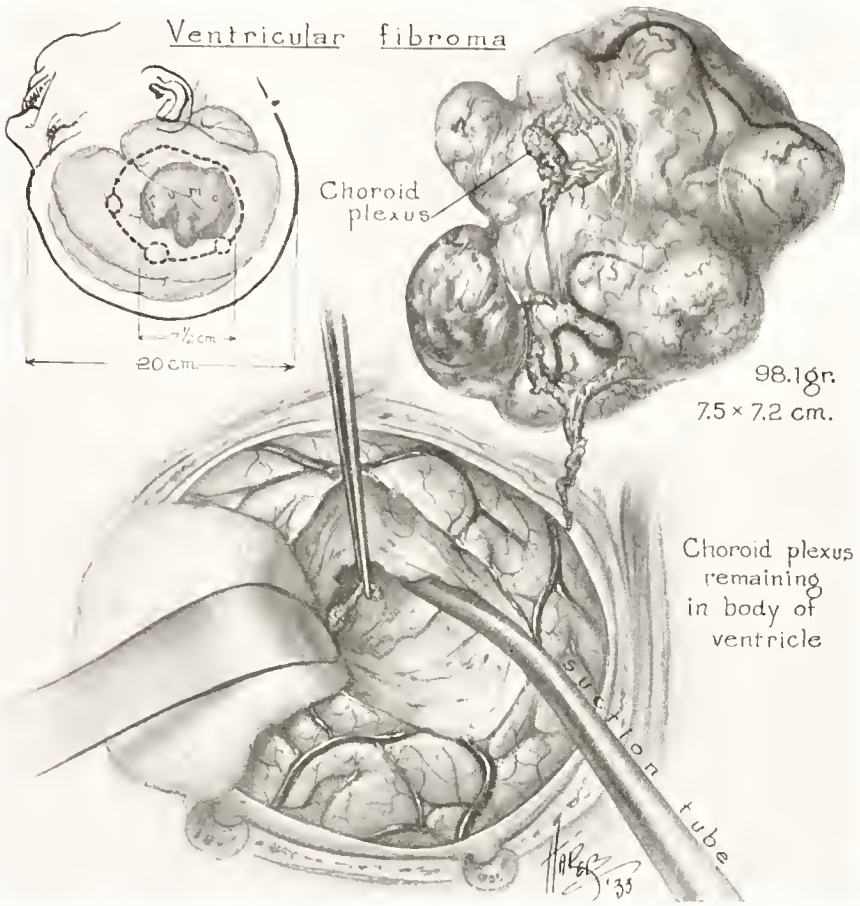

FIG. 72. Operative sketch showing position and character of the tumor (Case XIII). Note the choroid plexus attached at two widely separated points, between which the tumor doubtless arose.

The tumor weighed 98.1 grams. Necrotic brain tissue which was removed from the edges of the cortical defect weighed 13 grams. Cut section of the tumor showed a fairly uniform hard, white fibrous surface (Fig. 73).

Post-operative course (*Fig. 75*). The patient had perhaps a

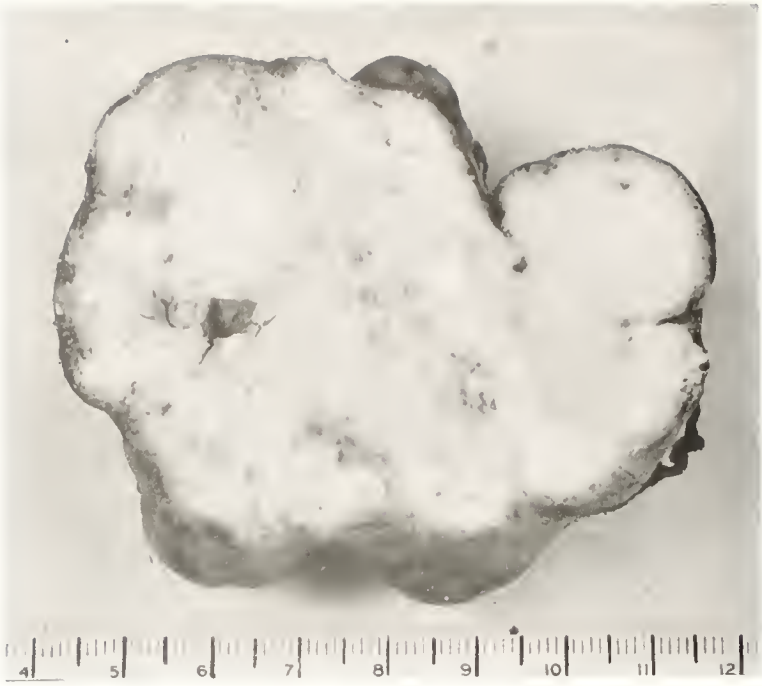

FIG. 73. Cut section of tumor (Case XIII) showing its firm fibrous texture

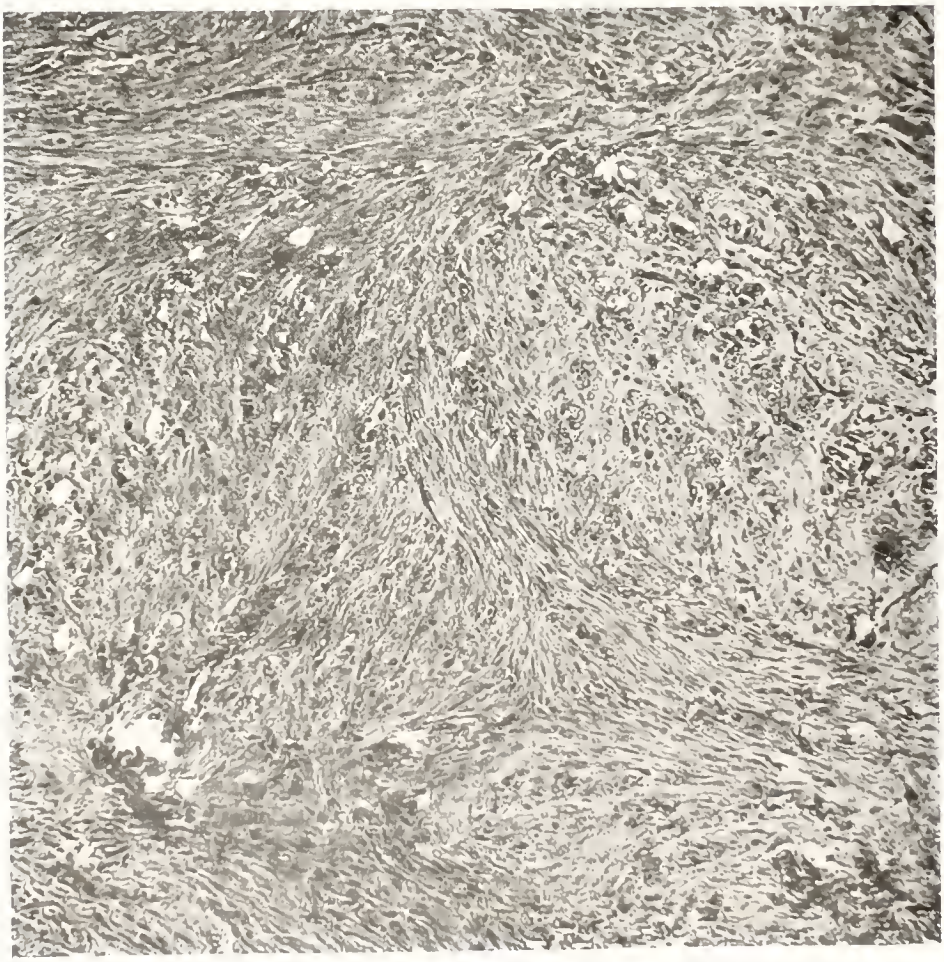

FIG. 74.1. Low power photomicrograph of tumor (Case XIII)

slight decrease of motor power on the left side after the operation, but this is questionable. At the time of this discharge

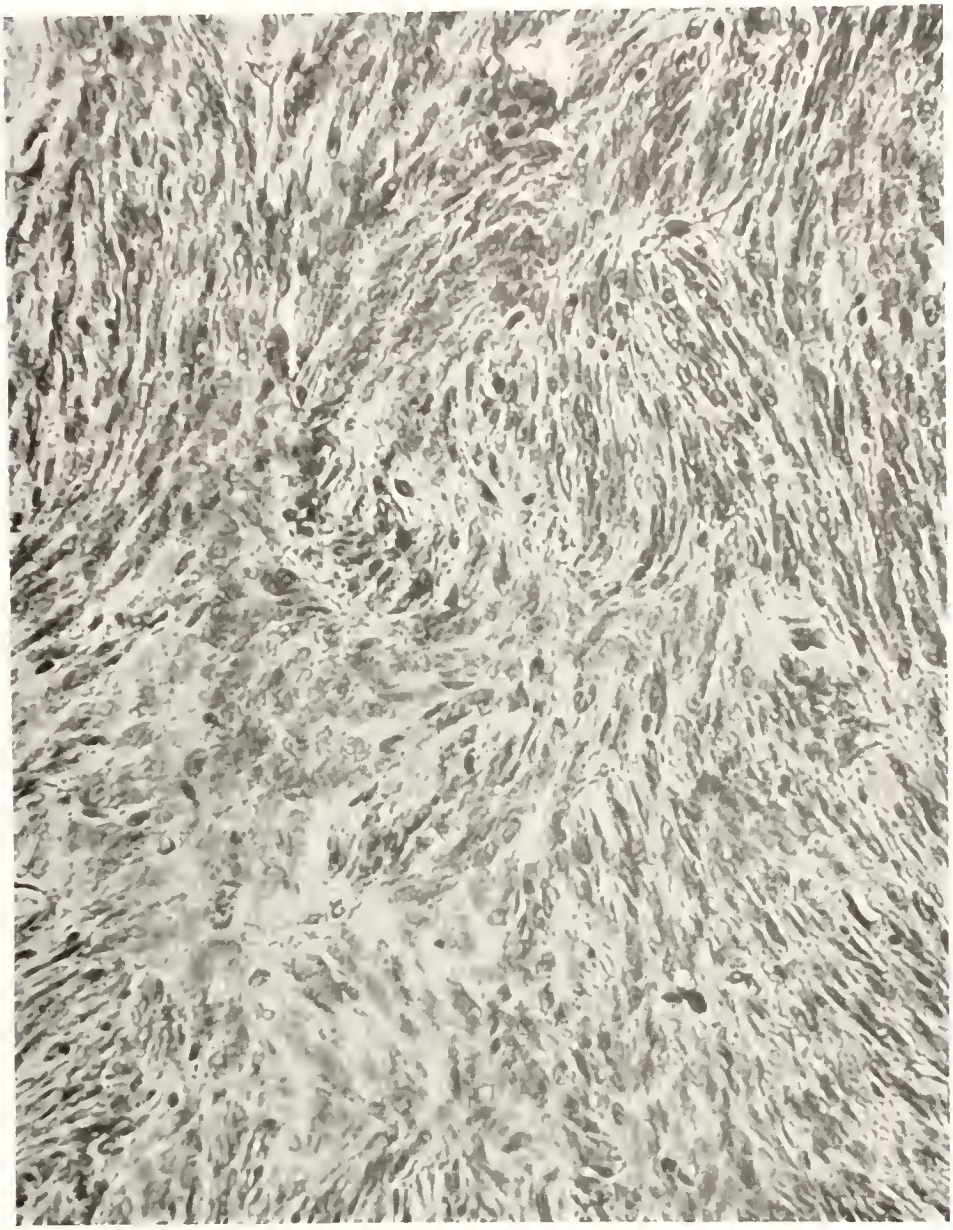

FIG. 74*B*. High power photomicrograph of tumor (Case XIII)

the function was better than when he was admitted. The sensory disturbance had improved and the astereognosis had entirely disappeared; the arm now "seemed to belong to him"

and no longer lost its position. The hemianopsia to the left remained unchanged and will doubtless be permanent. The audiometer curve shows some intensification of the auditory loss that existed before operation (Fig. 71).

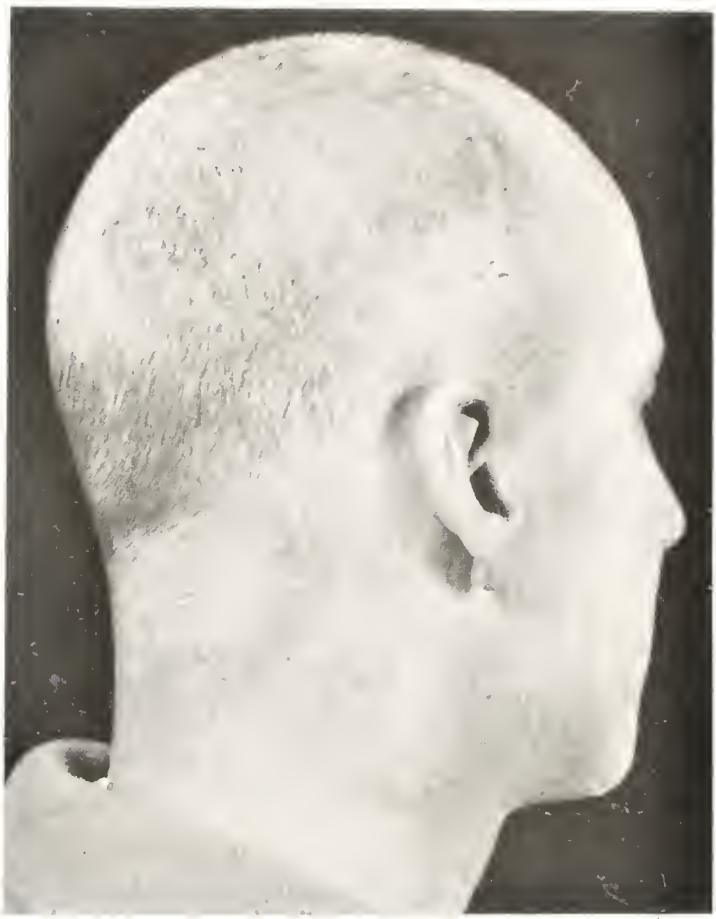

FIG. 73. Photograph of patient (Case XIII) at time of discharge

Microscopic description of the tumor. Fibrous tissue makes up the entire sections. It is closely packed with fibers arranged in every manner—parallel, tortuous, and in whorls (Figs. 74*A* and 74*B*). The tissue is fairly cellular; the nuclei are oval and elongated. There are no psammoma bodies and very little necrosis.

Diagnosis. Pure fibroma of choroid plexus.

Case XIV

S. A. Age 24. Admitted: August 24, 1933. Total enucleation of tumor. Discharged: September 10, 1933.

A very ill young man of 24 years was referred by Dr. Milford Levy, of Baltimore, with the diagnosis of a brain tumor.

Complaints. Headaches, failing vision, double vision and crossed eyes.

Family and past histories are negative.

Present illness. Began two months ago with morning headaches located behind both eyes. After moving about they usually disappeared. At the same time there was a dull ache in the back of the head and neck. The headache has continued up to the present time, but has not been very severe, and in the past few days it has been even less. Only on two occasions has he vomited.

At the time the headaches began, he noticed that his left eye turned inward, and synchronously double vision appeared. His vision has rapidly failed, more rapidly, however, in the left eye. At the present time he is blind. At no time has there been tinnitus or deafness. However, he has had the sensation of closure of the right ear, for which he was given some ear drops by a physician. Two weeks ago his tongue and lips felt numb, but he could not localize this disturbance to either side. There have been no sensory or motor disturbances, although on a few occasions his arms and legs felt as though they were asleep. For the past two weeks his neck has been quite stiff. Recently, his memory for recent events has been affected. During the past two weeks he has been quite drowsy. At no time has there been a history suggesting hemianopsia. There have been no convulsions.

Physical examination is negative.

Neurological examination. Patient is blind in both eyes. Papilloedema of eight diopters and numerous hemorrhages are present in both eyegrounds. I have never seen a higher swelling of the dises. Both external rectus muscles are weak.

Perhaps there is a slight ptosis and a little weakness of the superior rectus muscle on the left. Pupils are equal and react normally. A slight facial weakness of central type is present on the left, but no weakness of either arm or leg. There is no sensory disturbance and no astereognosis. The deep reflexes are sluggish on both sides, no ankle clonus, no Babinski. The Wassermann reaction from the blood is negative. The audiometer test is negative. X-rays of the head are negative.

Diagnosis. Intracranial tumor, not localizable. The left facial weakness suggests a tumor in the right hemisphere, but the asymmetry was not sufficiently defined to be regarded as unequivocal. For absolute localization ventriculography is necessary.

Ventriculography, August 25, 1933. The right lateral ventricle could not be reached through the posterior opening. From the left ventricle fluid spurted under tremendous pressure; 30 cc. of fluid were removed exhausting the ventricle, 20 cc. of air injected. The ventriculograms showed marked dislocation of the left lateral and third ventricles toward the left. The third ventricle was inclined obliquely at an angle of about forty-five degrees. The air did not reach the right lateral ventricle.

Diagnosis. Tumor in the right temporal or parietal lobe.

Operation, August 25, 1933. Immediately after the ventriculograms had been interpreted the patient was anesthetized with avertin. A large bone flap was turned down in the right parietal region. The dura was exceedingly tight and despite the release of fluid and air from the left ventricle the brain bulged severely when the dura was opened. There was no sign of tumor on the surface. The subarachnoid spaces were obliterated over all of the exposed brain. There was only one indication of an underlying tumor, i.e., the large Rolandic vein was pushed forward, thus indicating that the tumor lay posterior to it, but there were no surface changes in any of the convolutions to indicate the position of the underlying tumor. The anterior part of the temporal lobe was tapped and a small

amount of yellow, watery fluid escaped from the ventricle. It was evident, therefore, that the tumor did not extend to the tip of the temporal lobe. The ventricular needle was then inserted into the parietal lobe just mesial to the Sylvian vein and well posterior to the Rolandic vein. At a depth of 4 cm. a resistance was encountered and a small amount of brownish-black fluid—obviously the result of an old hemorrhage—escaped. A circular area of cortex, about 3 x 3 cm., was then excised from this region in order to expose the tumor. One could then see a small, pinkish, seemingly well encapsulated tumor. From this brief inspection it looked exactly like a fibroma of the choroid plexus, as shown in Cases XI and XII. The brain was then gently retracted anteriorly and the choroid plexus appeared upon, but apparently not attached to, the anterior border of the tumor. It was quickly enucleated with the finger. The tumor was very hard and appeared to be well encapsulated. A brisk hemorrhage followed, but was quickly controlled by a pack of wet cotton. When this was subsequently gradually withdrawn, several large veins were disclosed in the wall of the lateral ventricle and were coagulated with the cautery. It was now possible to see the entire choroid plexus, including the glomus. Apparently the plexus had been traumatized in the extirpation for several bleeding points had to be coagulated.

When the wound was dry one could look into the body, the posterior and descending horns of the ventricle. The tumor had filled the posterior horn and on its inferior wall a small area of brain tissue had been extirpated. It was apparent that this was the attachment of the growth. Through this small cerebral defect one could see the posterior margin of the tentorium. It was my impression at the time that the tumor probably arose from the subependymal layer and was an encapsulated ependymal glioma.

The patient's condition remained good throughout the operation. The blood pressure dropped to 80, and an intravenous injection of glucose was given. At the time closure was

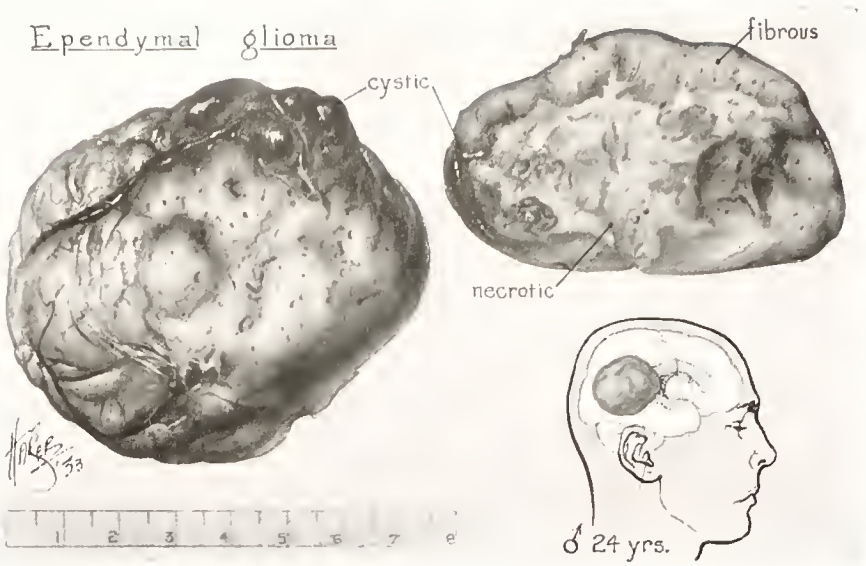

FIG. 76. Sketch showing gross appearance of tumor on surface and cut section, also position of tumor (Case XIV).

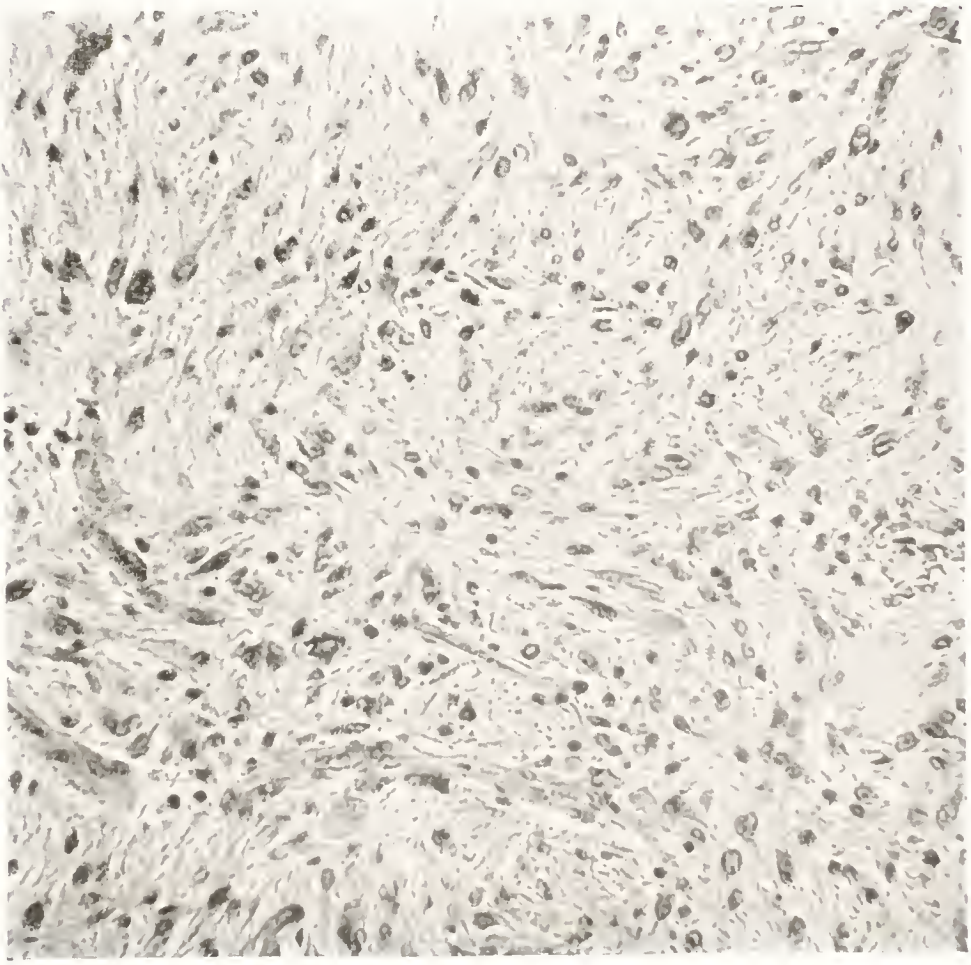

FIG. 77. Photomicrograph of tumor Case XIV

begun the brain still bulged through the dural defect and it was considered unsafe to close the dura and replace the bone flap; accordingly the dura was left open and the bone flap removed. The wound was closed without drainage.

The tumor weighed 56 grams (Fig. 76).

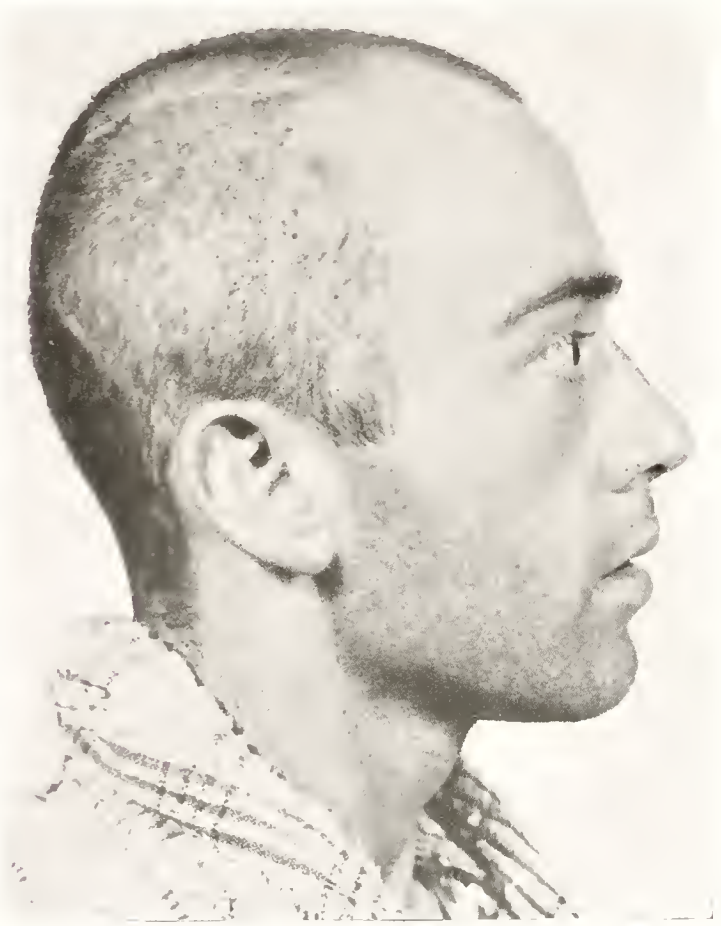

FIG. 78. Photograph of patient (Case XIV) at time of discharge

Post-operative note. The patient had complete hemiplegia on the left side. This was thought to be due to cerebral oedema as we felt quite certain that the cortical incision was too far back of the Rolandic area to cause permanent injury to the motor tracts. At the time of his discharge (Fig. 78) from the hospital the paralysis on the left side had greatly improved, so that he

could walk alone. Two weeks later he had only a slight limp. The arm and hand were somewhat more affected, but were steadily improving. He could then see fingers with the right eye, but the vision was restricted to the temporal field. The visual acuity was 8/100; no vision had returned in the left eye. There was no vision for color in the right eye.

Microscopic description of the tumor (Fig. 77). In many places the histological appearance is exactly like a glioma, but in many areas the closely compact fibrous strands, frequently in whirls, suggest a fibroma; the latter appearance is usually near the surface of the tumor, which has a sharply defined capsule. Necrosis is everywhere and in large areas. Glial stains show abundance of glial fibers, but only in patches. Much of the tumor contains no glial fibers.

Diagnosis. Ependymal fibroma or glioma.

Case XV

F. B. Age 11 years. Admitted: January 21, 1931. Total extirpation of tumor. Discharged: February 3, 1931.

Referred by the Department of Pediatrics, the Johns Hopkins Hospital, with the diagnosis of a cerebral hemorrhage probably from a brain tumor.

Past and family histories are entirely negative.

Present illness. On January 19, 1931, patient complained of frontal headache. Previous to that she had been perfectly well in every respect. That morning she ate no breakfast, but vomited several times during the day. In the afternoon her father thought she was not using her left hand normally; he asked her to pick up a handkerchief from a chair and she was unable to do so. She also dragged her left foot and had difficulty in walking. She was restless during the night. On the following day the headache was a little more severe. Her father thinks she used the left arm somewhat better than on the preceding day. On January 21, 1931 she was admitted to the hospital. Her physician at home took her temperature for two days and found no elevation. There had been no convulsions.

Physical examination. The patient is a pale, thin child, acutely ill; she is rational but very drowsy, and at times difficult to arouse. The eyes are kept closed except when she is stimulated, and even then it is difficult to persuade her to keep them open for more than a moment at a time. There is evidence of weakness of both internal rectus muscles. There is marked weakness of the entire left side of the body. The deep reflexes are hyperactive on the left side. The left corneal reflexes are sluggish. The abdominal reflexes are not obtainable. There is marked tenderness to pressure over the right side of the head, particularly the temporal-parietal region. Sensation is diminished throughout the entire left side, positive Kernig on both sides, positive Babinski on the left, but no ankle clonus, the neck is rigid. The eye-grounds show engorgement of the veins on the right and an early papilloedema on this side. There is perhaps slight blurring of the nasal side of the left disc. The visual fields show no deformation. The white count is 15,700. The differential count shows 71 per cent polymorphonuclear cells. Her pulse varied from 100 to 130, and her temperature 101° to 102° during the forty-eight hours following admission. On the day following her admission the patient was even more drowsy.

X-rays of the head were entirely negative.

Ventricular estimation, January 23, 1931. The left ventricle was punctured at the normal depth and fluid spurted under tremendous pressure; it was perfectly clear and contained one cell. The right ventricle could not be reached, but when the needle was withdrawn a drop or two of old, black blood dripped from the opening. This was absolutely definite indication of a cerebral hemorrhage and made it unnecessary to inject air for further proof either of a space-occupying lesion or of its position. The hemorrhage was encountered at a depth of about 8 cm.

Operation, January 23, 1931. Immediately after the above findings from the ventricular estimation an operation was performed in the right parietal-temporal region. A small

quantity of ether was used. The dura was very tight and the brain bulged markedly when the dura was opened. In the posterior region of the temporal lobe there were very suggestive signs of an underlying lesion. The convolutions were paler, wider, and flatter than normal, although the change was not great. There was no cerebrospinal fluid on the surface of this part of the brain and no old blood in the subarachnoid spaces. The nasal dilator was passed into the depths of the second temporal convolution, and at a depth of 5 cm. a blood clot was encountered. This was evacuated with the finger; a small firm nodule, about as large as a hazel nut, came out with the clot. Since it was delivered so easily, it could not be determined whether or not it had been attached. It was quite firm, smooth, and at the time was looked upon as a possible tumor nodule. Microscopic sections later showed this nodule to be a thrombus and not a tumor. The inferior portion of the temporal lobe was excised. It was necessary to remove this portion to get a view of the cavity; there remained only a small portion of the anterior part of the temporal lobe. Inspection of the cavity from which the hemorrhage had been delivered now showed that it extended backward towards the occipital lobe and upward towards the frontal lobe. The descending horn of the lateral ventricle was now open, but whether or not it had been opened during our manipulation was impossible to say. A careful inspection of the entire bed from which the hemorrhage was extirpated failed to reveal any further sign of tumor.

The wound was closed in the usual manner. The bone flap was replaced after a small decompression had been made. The patient made an uneventful recovery. Her paralysis rapidly cleared and she remained quite well until December 14, 1932, almost two years after her first attack. In the interim she had full use of her left leg and arm, although they were slightly weaker than the right leg and arm. There was no limp and no spasticity. There was, of course, a left homonymous hemianopsia presumably from the operative removal of the right

temporal lobe. Although it was difficult to believe that actual hemianopsia had not existed from the destructive effects of the hemorrhage, the visual fields tested roughly when she was very drowsy, were negative.

The patient went to bed in her usual good health, and at five o'clock in the morning she was awakened with a slight bifrontal headache and nausea; she called her mother and a short time later vomited. At this time she noticed that her left arm was weak, in fact it could be moved but very slightly. Later in the morning her left arm had become entirely useless and her left leg was almost functionless. She at once entered the neuro surgical service of the Johns Hopkins Hospital. She was entirely conscious and in no apparent physical distress. The left arm was totally paralyzed and but little movement was possible in the left leg; the left side of the face was also weak. There was partial hemianesthesia on the entire left side. The reflexes on the left side were exactly as at the time of the previous admission; the extraocular movements were normally performed. Her temperature ranged between 101° and 102°; her pulse, 134 and respirations, 26. On the following day there was some improvement in the power of the left arm and leg. Her headache was also slightly better. The decompression, which was now greatly reduced in size by the growth of bone on all sides, was full and tight.

Impression. It was evident that the patient had a recurrence of the old hemorrhage.

Operation, December 23, 1932. The old incision and bone flap were again turned down; the dura was not reopened, the defect in the region of the decompression being ample for our need throughout the operation. Superficially, the old cerebral defect was filled with fluid and strands of scar tissue. When these were removed a smooth membrane covering the inferior surface of the brain was encountered; it bulged and was a definitely bluish color. When opened an old blood clot escaped; the finger was inserted and a large hematoma, probably as large as a golf ball, was evacuated. The entire descending

horn of the ventricle was then in full view; the choroid plexus, including the glomus, was in full view. It was recalled that the ventricle was open at the last operation. The entire region was inspected and there was not the slightest evidence of a tumor, and the ventricle appeared to be entirely clear of cerebrospinal fluid. At one point, however, in the superior surface of the body of the ventricle, just anterior to the junction of the posterior horn, there was a small circular area of clot, about 1 cm. in diameter. As this was more carefully exposed it was seen to protrude into the parietal lobe through a narrow channel. As the channel from which the hematoma protruded was delivered, it was found that we were encountering a second hematoma of great size and within the inferior part of the parietal lobe. This hematoma when evacuated with the finger was about as large as the hemorrhage which had been evacuated from the descending horn. It was, therefore, a dumb-bell hemorrhage with a small neck which had burst through the roof of the body of the lateral ventricle. When this hemorrhage was evacuated the inferior surface of the parietal lobe, which seemed to be quite thin, was split with the scalpel. A blood clot was then wiped from the wall. At first no tumor could be seen but in the anterior-most part of this defect a hard nodular mass about as large as a small hickory nut was felt. The nasal dilator was then passed into this region and one could see the actual tumor. There were two large, rounded, bluish nodules which looked like distended veins, but were quite firm. One could also see that the tumor now projected into and filled the body of the lateral ventricle for perhaps 2.5 cm. from its posterior terminus (Fig. 79). The tumor had completely blocked the body of the ventricle and prevented the intraventricular hemorrhage from extending throughout the ventricular system and, therefore, reduced its effects to a local hemorrhage thereby saving the patient's life (Fig. 79).

The inspection of the tumor now revealed that the large artery and vein were crossing the mesial and anterior wall of

the lateral ventricle to reach the tumor. This blood supply had to be traced to the choroid plexus. Each vessel was as large as a slate pencil and made us exceedingly apprehensive of dangerous, perhaps uncontrollable bleeding, should they be

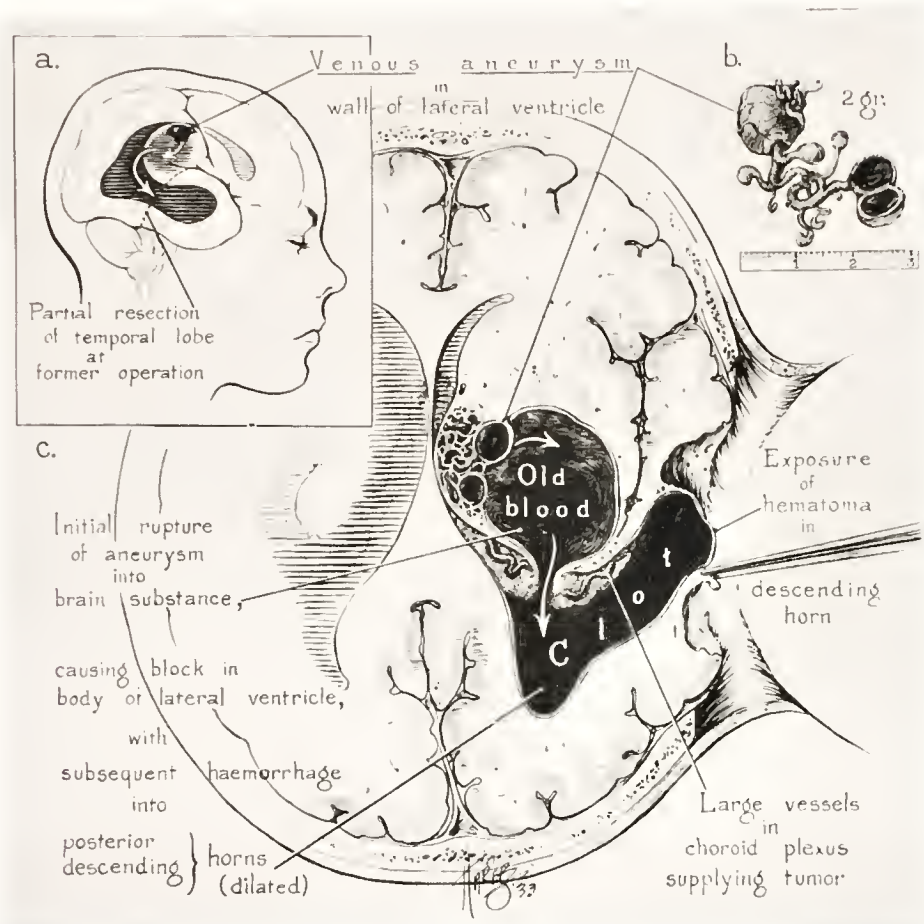

FIG. 79. Sketch from operative findings indicating the position of the angioma in the body of the right lateral ventricle (Case XIII). It will be noted that the hematoma fills the posterior and descending horns of the ventricle but not the remainder of the ventricular system, because the tumor had closed the body of the lateral ventricle. The inset shows the gross venous dilatation of the mass of the tumor after it had been dissected.

injured. Each vessel was doubly clipped and divided, and before division, the clips were cauterized to insure occlusion of the vessels. The finger was then gently passed around the tumor (its borders were very sharply defined) and the tumor

was enucleated in this manner with only slight bleeding. The bed from which the tumor was extirpated was exposed by suction of the fluid and a bleeding point picked up and cauterized. The bleeding was entirely stopped and the wound was perfectly dry. Inspection and palpation of the wound now revealed no further evidence of tumor, which unquestionably was an angioma.

After the removal of the growth cerebrospinal fluid backed up from the anterior part of the ventricle and was seen for the first time.

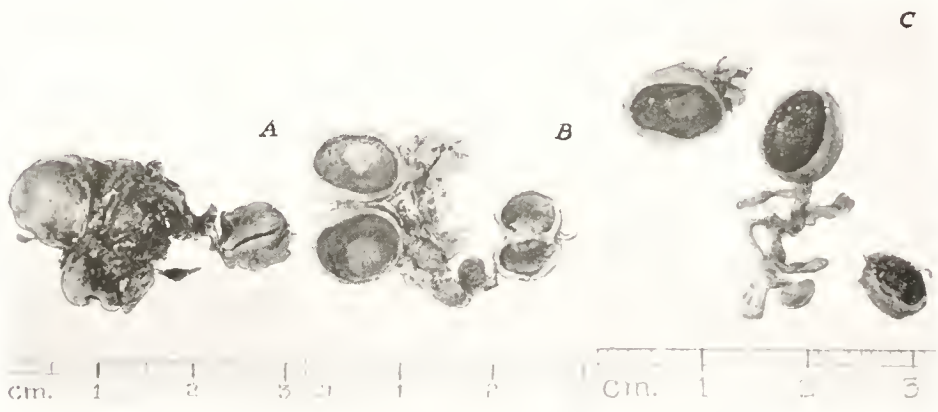

FIG. 80. Photographs of angiomatous mass removed from Case XIII.
A. Before dissection.
B. When thrombosed venous aneurysms were split.
C. Showing tortuous venous connections between the larger venous aneurysms.

It should be noted that the glomus of the choroid plexus was caught and removed because it dangled over the mouth of the body of the ventricle and prevented free access to the tumor in the ventricle. The removal was easy and was not accompanied by any bleeding. The large cerebral defect was filled with Ringer's solution, the bone flap replaced and wired; no drainage.

The weight of the tumor was 2.8 grams.

The patient made an uneventful recovery from the operation. A few days later motor function began to return. She was

discharged from the hospital two weeks later. A degree of motor deficiency of both the arm and leg persists (Fig. 83).

Gross appearance of tumor. There were two round, blue nodules, one as large as a pea, and the other about as large as

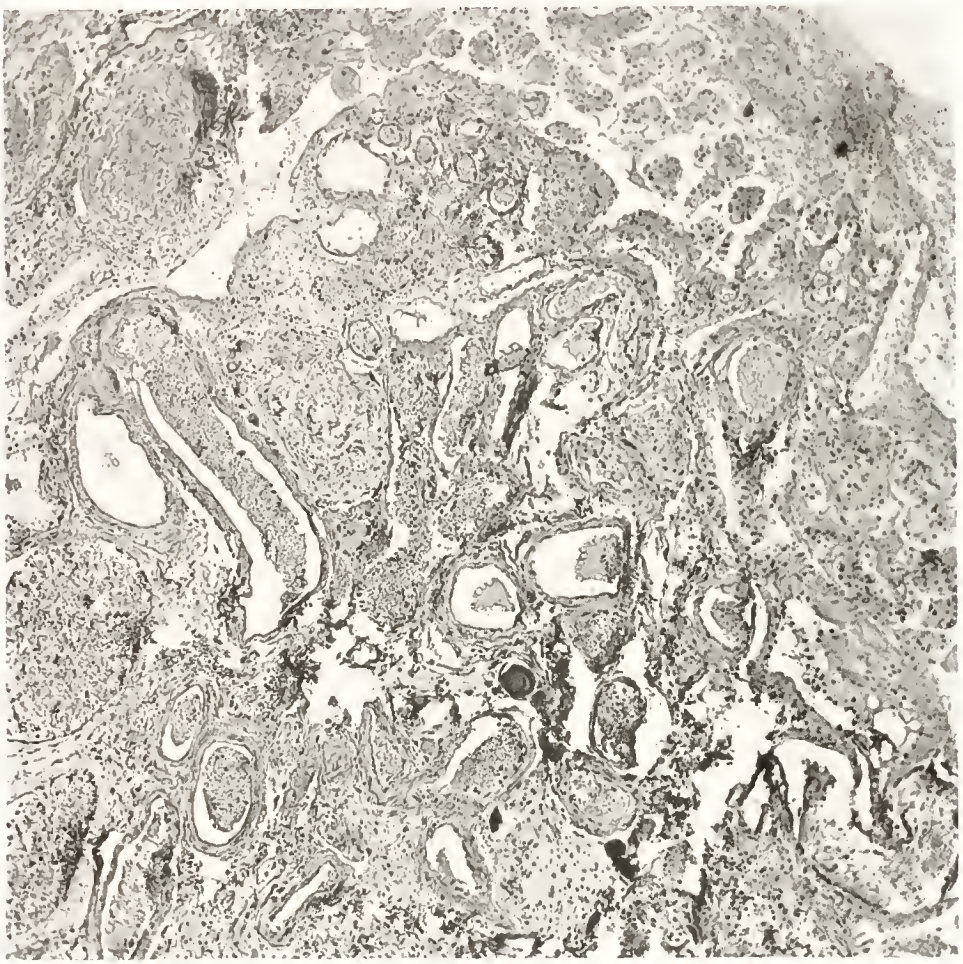

Fig. 81. Photomicrograph of angiomatous mass contained in the tumor removed from Case XIII. In the upper right hand corner one can see choroid plexus incorporated in the mass.

a hazel nut; they lay side by side and were held together by a bed of fibrous tissue, and in the extirpated specimen there was a small area of cerebral tissue (Fig. 80). When the nodules were opened each contained firm, laminated blood

clot. The wall of the vessel was quite thick but apparently not calcified.

Microscopic note. The outstanding feature of the section is the great number of blood vessels of different sizes closely matted together with fibrous tissue (Fig. 81). Usually the vessels have a fairly thick fibrous wall, but without differentiation of middle and outer coats; some, however, have little more

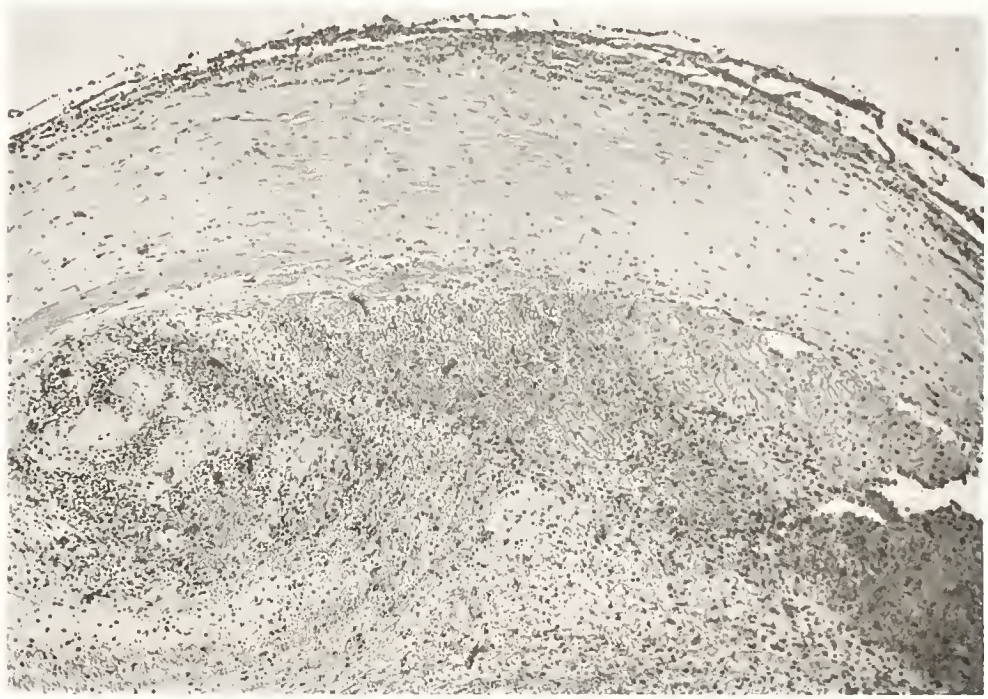

Fig. 82. Photomicrograph of wall and thrombus (Case XIII) of one of the venous aneurysms.

than an endothelial lining. Occasionally a tortuous, elastic layer suggestive of any artery is present.

Between many of the vessels are curious inclusions of columns of large epithelial cells, at times with the definite alveolar arrangement of choroid plexus (Fig. 81). In places these cells, which are much larger than normal choroid plexus, but unquestionably a derivative of that type of epithelium, are arranged in masses and disclose no tendency to alveolar formation. In

such places the regular cubical form of the cells is frequently lost and they look like large foam cells. Occasionally psammoma bodies are visible.

Section of one of the large vessels (seen with the naked eye) shows a wall of fibrous tissue of varying thickness. In some

FIG. 83. Photograph of patient (Case XV) ten months after operation

places the wall is very thin and practically replaced by thrombus (Fig. 82). There is no elastic layer in the intima and no endothelial lining, but everywhere attached to the inner part of the wall is a formed thrombus which completely fills the vessel.

Diagnosis—Venous aneurysm of choroid plexus.

CHAPTER V

ANALYSIS OF SIGNS AND SYMPTOMS OF ALL CASES (THIS SERIES AND THE LITERATURE)

AGE AND SEX

In twenty-seven of forty cases (67.5 per cent) the age of the patient was less than 31 years at the time of operation or necropsy. Nearly 50 per cent occurred before the twenty-first year and 25 per cent before the age of eleven. In three instances the tumor was present during the first few months of life. VanWagenen's case showed symptoms when two months old and was successfully operated upon in the third month. In Henning and Wagner's patient (1856) the tumor (probably a teratoma) was present at birth. Doubtless the cyst reported by Cayley and Brown was also present at birth for death resulted from it at the age of four months. Although there are several types of tumors in the lateral ventricle the large percentage with such an early time of appearance would appear to suggest that they were of congenital origin and arose from congenital "rests." There is perhaps some difference in the incidence of these tumors according to sex—twenty-two being in males and fifteen in females.

DURATION OF SYMPTOMS

The duration of symptoms is quite variable. Exactly half of thirty-four cases had symptoms less than one year. In three cases the symptoms had been present only a few weeks; and in ten, less than six months. On the other hand, several tumors caused symptoms of two to five years' duration, and in one instance, (Case VIII) symptoms persisted nine years. Although, in general, tumors that cause rapid progression of symptoms, both focal and general, are of the more malignant type, there are fortunately many exceptions.

Equally as rapid local symptoms may be due to cysts that develop even more quickly than the more malignant solid growths. Intracranial pressure may develop with much greater rapidity from a small benign tumor blocking some part of the ventricular system than from an enormous glioma that nearly compresses the ventricles. Tumors that arise within and occlude a ventricle and, therefore, cause fulminating intracranial pressure from hydrocephalus, are splendid examples of this fact. For example, in Cases II and XV of our series the tumors were very small but they produced the most rapid and fulminating symptoms, both focal and of intracranial pressure.

The only safe rule to follow with brain tumors is to assume that all are benign until proven otherwise; and they can be proven hopeless only by actual exposure of the lesion at operation. The long duration of symptoms in so many of these tumors is due to two conditions, (1) the tumors are so situated —usually in the anterior and posterior horns of the ventricle— that they do not cause hydrocephalus, or (2) the tumor, being encapsulated, acts as a ball-valve and only periodically occludes the ventricle.

FIRST SYMPTOM

From our series of fifteen tumors in the lateral ventricle, headache was the first symptom in thirteen cases. The two exceptions were Cases V and XIII. In Case V loss of vision appeared first; and despite the fact that the intracranial pressure was extreme and was responsible for the blindness, the headaches were slight. In Case XIII loss of libido was the first symptom. How to correlate this with the tumor is difficult to understand, but shortly afterwards vomiting attacks without headache appeared and persisted for a month before headaches ensued. It is equally difficult to explain this symptom, but it unquestionably was part of the illness. From the cases in the literature other disturbances occasionally preceded headaches: "fainting attacks" were the symptoms of onset in Hirsch's case; disturbance of speech and writing in

Davis and Cushing's Case III, and hemiplegia in Claude and Loyez's case. Among the very young children who doubtless had and could not register headache, enlargement of the head was first observed in VanWagenen's and Brüchanow's, vomiting in Ströber's and hemiplegia in our Case XV.

HEADACHE

As previously noted, headache may be due to the bulk of the tumor (plus that of the contiguous cerebral oedema when present), or the increased volume of fluid in the ventricles (hydrocephalus), or to both. It might well be expected that tumors confined to one side of the brain would cause headache to a much greater degree, at least, on that side. In six of our cases was this true (Cases II, IX, X, XII, XIII and XV), but only in three of these (Cases IX, XII and XIII) was the unilateral character of the headache strongly emphasized by the patient. It is clearly significant that in many instances the tumor was located on the side of the greater headache, but greater precision in localization of the tumor by the headache is scarcely possible. In five of these cases the headache was referred to the frontal region of the side of the tumor, but in only one was the tumor in the frontal part of the brain. In Case XII the headache was worse in the mastoid region, the tumor being in the parieto-occipital lobe—far distant from the tumor. In Paulian and Aricesco's case the headache was also strikingly unilateral; although the headache was in the left parietal region the tumor was in the anterior horn of the left lateral ventricle.

In our remaining nine cases the headache was frontal, temporal, occipital, and the vertex, always bilaterally equal or nearly so. In Hunziker's case the headache was so strictly unilateral that a diagnosis of migraine was made. Intensification of headache by bending over or sudden change of position was an outstanding complaint in Case II of our series. In none of the other patients were headaches increased in this way. Intensification of headache by coughing or straining was observed by one patient (Case XII).

TABLE I

WRITER'S SERIES OF BENIGN TUMORS IN THE LATERAL VENTRICLE AND CAUSING SYMPTOMS

CASE REPORTED	DATE OF OPERATION	AGE	SEX	DURATION OF SYMPTOMS	FIRST SYMPTOM	INTERMITTENT ATTACKS	HEADACHE	VOMITING	DIPLOPIA	DIZZINESS	BILATERAL PAPILLOEDEMA	VISUAL CHANGES	EXTRAOCULAR PALSIES	STAGGERING GAIT	ROMBERG	ATAXIA	MENTAL CHANGES	POLYURIA AND POLYDIPSIA	ENDOCRINE AND VASOMOTOR DISTURBANCES	DISTURBANCE OF SPEECH	MOTOR OR SENSORY DISTURBANCES	CONVULSIONS	CHARACTER OF CONVULSIONS	REFLEXES	TINNITUS	AUDIOMETER TEST	OTHER SIGNS AND SYMPTOMS	X-RAY OF HEAD	TENTATIVE DIAGNOSIS BEFORE VENTRICULOGRAPHY	TUMOR LOCALIZED BY	TUMOR DISCLOSED BY: Necropsy	TUMOR DISCLOSED BY: Operation	TYPE OF OPERATION	RESULT	TIME ELAPSED SINCE OPERATION	SIZE OF TUMOR	CHARACTER OF TUMOR	CYSTS	MICROSCOPIC	HYDROCEPHALUS	REMARKS
I. W. B.	1918	23	M	4 years	Headache; diplopia 5 weeks, disappeared, never returned	+	+ All over head	+ Very frequent	+		+	Marked loss, color fields almost gone	No	+	+	+	Loss of memory	No	None	No		No (?)	Was unconscious 4 days, then returned to normal	Normal			Loss of smell and taste; nystagmus; possible uncinate attacks	Negative	Cerebellar tumor	Ventriculography		+	Enucleation of tumor	Well	15 years	Small orange	Ependymal fibroma	Encapsulated, soft, nodular	Loose fibrous tissue; occasional glial fibers	Moderate grade	Diplopia was one of the first symptoms, lasted five weeks, disappeared and never returned. This was first brain tumor localized by ventriculography
II. C. M.	1920	23	M.	4 months	Headache		+ More in right frontal region	+	+ Transient	No	+	Marked loss	No	+ One attack	Negative	No	None	No	None	No	Partial hemiplegia followed convulsion	+ Only in hospital	Unilateral	Normal	+ Buzzing in right ear		Bending over intensifies headache	Negative	Unlocalized tumor of the brain	Ventriculography		+	Enucleation	Well	12 years	9 grams	Ependymal fibroma	Encapsulated, round, hard	Fibrous tissue; occasional glial fibers	Only in posterior and descending horns of right ventricle	
III. H. F.	1920	12	M.	2 years	Headache	+	+ Top of head	+	+	+	+ Slight	Blind	+ Slight	+	+ To right	Suggestive	None	No	None	No	No	None	Knee kick increased on right	+ Buzzing in both ears		Loss of smell; tremor in both hands	Convolutional atrophy and separation of sutures	Cerebellar tumor	Cerebellar operation		+	Cerebellar	Dead		Small orange	Ependymal fibroma	Hard, nodular, encapsulated	Specimen lost	+ Both ventricles greatly enlarged	Tumor was found at cerebellar operation but was beyond reach. Ventriculography should have been used, and would have made the correct localization
IV. S. J.	1925	30	M.	6 months	Headache	+	+ Frontal and general	+		No	+	Blurred during attacks only	No	No	No	No	None	No	None	No	None	None		Normal			Stiffness of neck	Negative	Unlocalizable tumor	Ventriculography		+	Resection of frontal lobe and enucleation of tumor	Dead		9 5 grams	Round cell tumor—ependymal ?	Hard reddish-brown, encapsulated	Closely packed round cells	+ Unilateral	
V. W. S. R.	1930	38	M.	15 months	Loss of vision	+	+ Vertex	+	+	No	+	Almost blind	No	No	No	No	None	No	None	No	None	No		Normal	No	Loss of high tones in both ears		Negative	Unlocalizable tumor	Ventriculography		+	Resection of U-shaped area of right frontal lobe; enucleation of tumor	Well	2½ years	7 grams	Ependymal glioma or fibroma	Hard, white, dumb-bell	Mixture of glial and fibrous tissue	+ Bilateral but ventricles not in communication	Much glial tissue in sections
VI. R. A.	1931	40	M.	4 years	Headache and nervousness	+	+ Bifrontal and occipital	+	No but blind in left eye 2 years		+	Blind in left eye (tumor is on right)	No	No	No	No	Marked personality changes. Forgetful	+ Formerly nycturia 4–5 per night; now 1–2	None	No	Some subjective weakness left leg; no objective change	+	Petit and grand mal; no focal signs	Normal	No	Slight loss of high tones in both ears	Patient has had pulmonary tuberculosis	Negative	Unlocalizable tumor	Ventriculography		+	Removal of tumor; resection of right frontal lobe	Well	2 years	14 9 grams	Ependymal fibroma	Hard, white, encapsulated; also filled third ventricle	Specimen lost	+ High grade	Ventriculogram showed filling defect in right lateral ventricle. Bilateral hydrocephalus because tumor also filled third ventricle. Septum pellucidum atrophied
VII. L. D.	1930	19	F.	2 years	Headache	+	+ Occipital	+	No		+	Blurring also transient blind spells	No	No	No	No	None	No	None	No	None	+	No focal signs	Normal		Negative	Exhaustion and nervousness which she attributes to convulsions	Negative	Unlocalizable tumor	Ventriculography		+	Resection of right frontal lobe; enucleation of tumor	Well	2½ years	47.7 grams	Ependymal fibroma	Very hard, nodular, solid growth	Fibrous tissue and areas like angioma	None	
VIII. B. W.	1928	33	M.	9 years	Headache	+	+ Occipital	No	No	+	+	Marked loss of vision	No	+	No	No	None	No	None	No	Numbness and tingling right arm and leg; no objective change	None		Normal	+ Thumping noises in right (contralateral ear)	Almost total deafness in left ear (not due to tumor)	Thumping noises in ear induced by noise; shaking knees	Negative	Unlocalizable tumor	Ventriculography		+	Enucleation; exposure by separating left frontal lobe	Well	4½ years	18 grams	Round cell ependymal tumor	Pedunculated, fairly firm	Closely packed round cells—like Case IV	+ Bilateral but no communication between lateral ventricles	Tumor projected through foramen of Monro into the third ventricle. Ventriculography showed filling defect in left lateral ventricle
IX. E. E. S.	1931	9	F.	5 months	Headache	+	+ Left frontal always	+	No	No	+	None	No	No	No	No	None	No	None	No	None	No		Normal	No		Drowsiness	Convolutional atrophy	Unlocalizable tumor	Ventriculography		+	Resection of left frontal lobe; enucleation of tumor	Dead		13 grams	Embryonal ependymal tumor	Soft, fluffy, pedunculated, reddish brown	Tumor made up of large unidentified cells of varying shape	+ Unilateral; state of opposite ventricle not determined	Tumor showed beautiful filling defect in anterior horn of left ventricle (ventriculography). Weight of excised frontal lobe was 66 grams
X. J. M.	1927	14	F.	5 years	Headache	+	+ Frontal, more right	+	No	No	Bilateral primary atrophy; veins full and tortuous	Almost blind	No	No	No	No	None	No	None	Thick speech on one occasion	Numbness of the right face and tongue; bilateral spasticity, more left	No		Left Babinski, ankle clonus, knee kick increased	No		Periods of drowsiness; local bulging of skull on right with crepitation to palpating finger; numbness left side of face in attacks	Convolutional atrophy	Right cerebral tumor	Ventricular estimation; cyst tapped		+	Removal solid tumor	Well	6 years	60 grams + large cyst	Adenoma of choroid plexus	Solid and cystic	Like choroid plexus	+ On left only	
XI. F. L.	1930	12	F.	1 year	Headache	+	+	+	+ Only once	+ Postural	+	Blurring more right; blind spells	No	No	No	No	None	No	None	No	None	No		Normal	No		Dizziness on changing position, but recently constant; asymetry of head	Negative	Unlocalizable tumor	Ventriculography		+	Resection cercular area left parietal cortex; enucleation of tumor	Well	2½ years	124 grams	Pure fibroma of choroid plexus	Hard, round smooth, elastic	Compact fibrous tissue	+ Only in descending horn of left ventricle	
XII. L. A	1933	35	M.	5 months	Headache	Slightly +	+ General and to right mastoid	No	No	No	+ 6 diopters	Loss of acuity and *right* hemianopsia for colors only (tumor is on *right*)	No	No	No	No	None	No	None	No	Complains of clumsiness of *right* leg; no objective changes (tumor is on *right*)	No		Normal	+ In left (contralateral ear)	Tremendous abrupt loss of high tones on both sides	Possible uncinate symptoms; headaches worse after coughing or straining; loss of vibratory sense in *left* ankle and knee	Negative	Probably *left* cerebral	Ventriculography		+	Removal of tumor; excision parietal cortex	Well	2 months	95 grams	Pure fibroma of choroid plexus	Hard, solid, elastic, smooth, oval	Compact fibrous tissue	+	Ventriculograms showed filling defect in posterior half of third ventricle where obstruction to ventricular system occured. Although patient had complained of a clumsiness in the *right* leg for 5 months (one of earliest symptoms), no objective loss of function could be disclosed, except the loss of vibratory sense in the *left* ankle and knee
XIII. J. W. F.	1933	48	M	13 months	Loss of libido; then vomiting, headache later	+	+ Back of eyes, more on right	+	No	No	Slight on right	Left homonymous hemianopsia	No	+	No	No	Difficulty in making legal decisions	No	Loss of libido	No	Slight motor and greater sensory loss on left	No		Deep reflexes increased on left; positive left Oppenheim	No	Loss of high tones in both ears	Listless; asterognosis left hand; loss of sense of position of fingers and toes on left	Negative	Right cerebral tumor, probably in perietal lobe	Neurological signs		+	Removal of tumor	Well	3 weeks	98.1 grams	Pure fibroma of choroid plexus	Encapsulated, nodular, hard	Compact fibrous tissue	On left (contralateral side)	
XIV. S. A.	1933	24	M	2 months	Headache	No	+ Over eyes and back of head and neck	+	+	No	+ 8 diopters	Blind	+ Bilateral VI	No	No	No	Memory for recent events affected	No	No	No	Slight left facial weakness	No		Sluggish but equal	No	Negative	Sensation of closure of right ear; stiffness of neck	Negative	Perhaps right cerebral tumor	Ventriculography		+	Removal of tumor	Well	1 month	58 grams	Ependymal glioma	Encapsulated, smooth, hard	Fibrous tissue with many patches of glia	On left (contralateral side)	
XV. F. B.	1932	13	F.	3 days and 2 years	Headache	+	+ Frontal and right parietal	+	+	No	Unilateral on side of tumor	None	+	Possibly	No	No	None	No	None	No	Hemiplegia left	No		Increase on left; Babinski left	No		Signs of intracranial pressure due to sudden hemorrhage	Negative	Right cerebral tumor with hemorrhage	Ventricular estimation		+	Removal of tumor	Well	6 months	2 8 grams	Venous angioma and venous aneurysm	Series of venous pouches matted together into a tumor	Venous walls and fibrous tissue; also inclusion of choroid plexus epithelium	+ Localized	At an earlier operation (2 years ago) hematoma was evacuated but tumor not disclosed

VOMITING

Vomiting usually closely parallels headache, but not necessarily so, as is shown in Ströber's case and in Case XIII of my series, where the vomiting antedated the headache by a month. It was present in all except two of our cases (VIII and XII). Usually vomiting comes when the headache is at its peak; and after vomiting, the headache is usually relieved. Vomiting may or may not be projectile. Of far greater significance is its relationship to headache.

INTERMITTENT ATTACKS

In my recent publication on tumors in the third ventricle, the intermittent character of the attacks was emphasized. Since intermittent intracranial pressure is due to periodic obstruction of the ventricular system, it is apparent that tumors in the lateral ventricle should afford excellent examples of this kind. In thirteen of our cases, intermittent attacks were emphasized by the patients. In the remaining cases (II and XIV) no mention of periodicity is made. In at least two cases from the literature, those of Barré and of Metzger and Lydston, periodic attacks are described. Only in the case of Davis and Cushing (III) is the absence of periodic attacks specifically denied.

DIPLOPIA AND EXTRA-OCULAR PALSIES

Diplopia was present in eight of our cases; it was transient in at least two of these, and was accompanied by extra-ocular palsies in only four. Only in Cases XIV and XV were the extra-ocular palsies severe. The absence of these subjective and objective disturbances in so many cases is perhaps surprising. It is doubtless due to the distance of the tumor from the third, fourth, and sixth nerves where they cross to the dural covering of the cavernous sinus; but the frequency with which these nerves are involved in all brain tumors causing intracranial pressure would hardly lead one to suggest this degree of immunity from tumors in the lateral ventricle. It is

interesting that in Case I diplopia was one of the earliest symptoms. It lasted two weeks, then disappeared and never again returned.

DIZZINESS

Dizziness was present in four of fourteen cases. In two instances the patients felt unsteady much of the time, as if drunk. In two other cases spells of dizziness lasting only a few moments were brought on by movement of the head or changes in posture; in each there was a loss of vision during the period of dizziness. In three cases from the literature the dizziness is noted, Hunziker, David and Cushing, and Jumentié, Olivier and Leclaire. Though perhaps less intense, dizziness on change of posture is essentially similar to that described in tumors in the third ventricle.

BILATERAL PAPILLOEDEMA

Papilloedema was present in all but one (Case X) of our sixteen cases, but was of very low grade in three cases, in two of which (Cases XIII and XV) it was unilateral and on the side of the tumor. In Case X papilloedema was absent, the discs being sharply defined and atrophic, but the retinal veins were quite full and tortuous. The incidence of papilloedema is essentially the same as for tumors giving a similar degree of pressure and located elsewhere in the brain. In two cases from the literature—Hirsch and Davis and Cushing—papilloedema was absent despite the existence of intracranial pressure of high grade. Although by far the most important sign of intracranial pressure, its absence can never preclude the existence of a tumor.

VISUAL CHANGES

Loss of vision of varying degree was present in most cases; in many there was blindness. Attacks in which vision was blurred or lost temporarily occurred in several patients and doubtless was a forerunner of total blindness which would have

followed shortly. Usually the initial loss is on the side of the tumor, but it may be greater in the opposite eye. However, in the end the vision in the two eyes is usually not greatly different. In one case the loss of vision was much greater on the side opposite the tumor.

Loss of vision is, with perhaps one exception (Case X), due solely to the effects of intracranial pressure. It is quite possible that the loss in this instance is of similar origin, but it was our impression that the tumor may have compressed the nerves directly—though seemingly well above them—and caused primary atrophy.

Homonymous hemianopsia for form and color was present in Case XIII and was an important localizing sign. In Case XII we might well have been badly misled by the perimetric examinations. This patient, whose tumor was in the right hemisphere, had homonymous hemianopsia, for colors only, to the right. This disappeared after removal of the tumor.

STAGGERING GAIT, ROMBERG, ATAXIA

These subjective and objective findings were present in some degree in five of our cases. Neurologists and surgeons have learned to be distrustful of these findings, especially if equivocal and not consistent. But since they remain the most important signs of localization to the posterior cranial fossa, it is not surprising that they still mislead. They were responsible for a cerebellar operation in Case III of our series and Case II of Davis and Cushing. In none of our cases were the staggering and ataxia outstanding signs; usually they were periodic and transient, and were more in evidence during exacerbations of intracranial pressure. It will also be recalled that these signs are frequently no less pronounced in the presence of the more favorable tumors of the cerebellum.

MENTAL CHANGES

Four of our patients showed slight mental disturbances. In Cases I and XIV there was some loss of memory; in Case VI,

forgetfulness and marked personality changes; and in Case XIII, difficulty in making legal decisions. The number of instances of mental disturbance among the cases reported in the literature is much greater. For example, Audry's patient was alternately excited and depressed; in Collin's case there were progressive mental changes; Hunziker's patient was depressed and forgetful; in Hart's case there was loss of memory and in the case reported by Jumentié, Olivier and Leclaire forgetfulness, irritability and alternating periods of timidity and defiance dominated the illness. Of the tumors causing mental symptoms seven are located in the right lateral ventricle and two (Audry and Hunziker) in the left. The changes enumerated are like those associated with intracranial pressure of high grade and are probably not at all significant of the tumor's location—even to the side of the brain involved. From these cases there appears to be no striking difference between the changes in the right and left sided tumors.

POLYURIA, POLYDIPSIA, ENDOCRINE AND VASOMOTOR DISTURBANCES

These disturbances have been present in only one case (VI) of our series, and in none from the literature. In Case VI the tumor also filled the third ventricle. In a review of tumors in the *third* ventricle there was a single instance—and it was outstanding—of an endocrine disorder. The patient, a young girl, was a dwarf who had some menstrual irregularity and perhaps some hirsutism. It was distinctly our impression that the character of the tumor and not its location was responsible for her retarded somatic development. In none of the benign tumors in the third ventricle was there any evidence of polyuria or polydipsia. Transient vasomotor changes manifested as sharply defined areas of reddening of the skin were reported in some of the tumors in the third ventricle. These have not been observed in any of the tumors in the lateral ventricle. However, the absence of these changes cannot be

stressed very strongly because it is quite possible that they might not have been reported by the patient and might have been overlooked or regarded as without significance among the earlier cases.

DISTURBANCE OF SPEECH

Aphasia as a localizing sign needs no comment. However, in none of our cases was there either aphasia or paraphasia. The absence of aphasia when a right sided hemiplegia develops in a right handed person may well be significant of a very deeply lying tumor, but there was no such disassociation in our series. The only possible disturbance of this type recorded in our cases was "thickness of speech" during attacks of increased intracranial pressure, and in this case (X) the tumor was on the right side.

MOTOR AND SENSORY DISTURBANCES

The most important localizing sign produced by tumors in the lateral ventricle are hemiplegia and hemianesthesia. Well developed hemiplegia was present in two of our cases (II and XV), and paresis of the entire side in one other (XIII). In three cases there was contralateral facial weakness without involvement of the corresponding arm or leg. The prior and greater involvement of the face is significant because it suggests a lesion at a greater distance from the midline. In Case II the paralysis came suddenly after a convulsion, was complete and persisted until the time of operation a few days later. The rapidity with which hemiplegia develops is doubtless significant, and perhaps also, in some instances, its transient character. It is perhaps suggestive of a deeply situated tumor—though one could scarcely infer more—when the hemiplegia develops suddenly, because it affects the internal capsule. When the cortical centers are involved by a tumor the motor loss is gradually progressive and one member is paralyzed for some time before a contiguous part on the same side. The

very sudden involvement of the internal capsule (in Case II) was probably due to the cerebral oedema (incident to the convulsion) about a tightly packed tumor lying directly against the internal capsule. In two cases (I and X) facial numbness developed suddenly on the side of the tumor and later disappeared. When anesthesia is restricted to the face, and particularly when transient, it is very strong evidence that the peripheral nerve (in this case the Gasserian ganglion) is at fault and not the cerebral sensory tracts. In Case VIII there was subjective numbness and tingling of the right arm and leg; in Case VI, subjective weakness of the left leg, but no corresponding objective evidence supported the complaint in either instance. The danger of placing too much emphasis on such subjective and unsupported complaints is evident in Case XII. This patient complained of weakness of the right leg, but his tumor was on the right side of the brain.

Although hemiplegia is a most valuable objective sign in localizing the tumor to a hemisphere, it should again be emphasized that for the surgeon far greater precision in localization is necessary. The great depth of intraventricular tumors would make their disclosure at operation very difficult, uncertain and usually impossible, except for the positive information afforded by ventriculography.

It is also worthy of note that the motor changes may be bilateral, due to the fact that the ventricles are not far apart and extension across the dividing line is possible. In Case X there were bilateral spasticity and reflex changes, although before becoming bedfast she had not complained of motor weakness. The spasticity was greater on the side opposite the tumor. In Jacarelli's case there was bilateral paralysis, but more marked on the side contralateral to the tumor. Seven additional cases from the literature show some degree of hemiplegia or hemianaesthesia (see chart). In case XII there was loss of vibratory sense and sense of position in the left ankle and knee and in Case XIII in the entire left side; astereognosis was also a striking objective finding.

CONVULSIONS AND THEIR CHARACTER

Only three of our cases (20 per cent) had convulsions. In Case II there was but a single convulsion which was unilateral and hemiplegia followed in its wake. Case VII in addition to convulsions had "rigid spells" involving the entire body and without loss of consciousness. In another patient the convulsions were clonic and generalized but at no time was there a suggestion of focal character to the convulsion. It is possible that there may have been uncinate attacks but without convulsions in Cases I and XII.

In seven of the cases in the literature convulsions of different types were described. In the reports of Hirsch and of Davis and Cushing (Case III) there were Jacksonian attacks with a definite "march." Unilateral convulsions were described by Jumentié, Olivier and Leclaire. It is evident, therefore, that any type of convulsion may result from tumors located in the lateral ventricles.

REFLEXES

Alterations in the deep reflexes are in evidence when there is loss of motor function; and at times the reflex changes antedate the motor loss. In Case III the reflexes were increased on the right, although motor changes were absent. In Cases X and XV there were unilaterally increased reflexes and positive Babinski. Ankle clonus on the left was also present in Case X. In eleven of the cases in this series the reflexes were normal.

TINNITUS

The localizing significance of tinnitus is not known. When a persisting *pain* in one ear is caused by a brain tumor the lesion is usually in or near the temporal lobe of the same side. But there is no such assurance of localization with tinnitus, which, of course, has many causes other than tumors; in fact, tumors are only the occasional cause. Tinnitus was present in four of our fifteen cases. In Case II the tinnitus was on the side of

the tumor. In Case III there was tinnitus in both ears; and in Cases VIII and XII tinnitus was only in the contralateral ear. It is perhaps worthy of note that in Case VIII there was, from causes unknown, almost total deafness on the side of the tumor. Certainly the tumor was not the cause of the deafness, but whether the deafness precluded tinnitus in this ear is impossible to say. In one instance, therefore, tinnitus was on the side of the tumor, in two it was on the opposite side and in one it was bilateral. An interesting but inexplainable symptom was noted in Case XIV. For sometime he complained of a sensation "that the right ear (side of tumor) felt full and as if closed." It was sufficiently disturbing to visit a physician for relief.

OTHER SIGNS AND SYMPTOMS

In young children the shape of the head will at times betray the existence of an underlying tumor; it may, in fact, be the only sign of localization. The mere enlargement of the head together with Macewen's sign (cracked-pot) will disclose the presence of intracranial pressure as in Brüchanow's case. In Case XI, a girl of twelve years, the parietal region bulged on the side of the tumor, and in addition the distance from the ear to the midsagittal line was much greater on that side. This was observed, however, only after the head was shaved and the tumor had been localized by ventriculography. The existence of cranial assymmetry at this relatively late age must imply the presence of the tumor in the early years of life when the skull was plastic. Similar localized enlargement of the head over the tumor is reported by Cayley and Brown. Localized tenderness over the tumor was found in the cases of Hunziker and Hirsch. A crepitant sensation of thin paper-like bone was detected on palpation of the localized bulge over the tumor in Case X.

Loss of taste and smell is recorded in Case I and of smell only in Case III, but there is no proof that either loss is related to the tumor. Tremor of both hands was present in Ströber's

case, that of Davis and Cushing (III) and our Case III. Stiffness of the neck was a conspicuous symptom in Case IV. Though an important cerebellar sign, one is never justified in making a diagnosis of a tumor in the posterior fossa on the basis of cervical rigidity. It is by no means uncommon to find tumors elsewhere in the cerebral hemispheres, and especially in the region of the pineal and third ventricle giving this sign.

Recurring attacks of drowsiness were noted by only one of our patients (Case X). They seemed to occur just before the severe headaches were due. More or less constant drowsiness and lethargy became pronounced symptoms in Case XIII.

Loss of sphincter control of the bladder and rectum developed in Case IV of our series; a similar disturbance is reported by Jumentié, Olivier and Leclaire. Loss of libido was the first symptom in Case XIII and antedated all other symptoms by a few weeks.

AUDIOMETER TEST

A search for changes in the audiometer curves—i.e., loss of high tones—that might be significant in localization has yielded but one unequivocal example (Case XII); here the changes were most striking. But it is worthy of note that the tumors in Cases XI, XIII and XIV were seemingly identically placed and of approximately the same size and yet the audiometer curves were practically normal. Much less pronounced bilateral loss of high tones were found in Cases V and VI, but the patients had not complained of subjective loss of hearing. In analyzing the audiometer curves of primary tumors in the third ventricle, sharply defined *bilateral* loss of high tones occurred in a few cases and led us to wonder if the acoustic defect of this form might not localize the lesion. The hope was stimulated the more by the fact that upon such an audiometer curve a tumor in the third ventricle was predicted and found at operation. Recently I saw identical bilateral acoustic curves in a patient who had a central cerebellar cyst that extended well

anteriorly and compressed the midbrain. We have also occasionally seen similar curves in other intracranial lesions that were not tumors and were without known localization. The picture, therefore, is not pathognomonic of a tumor, but I still believe that it indicates a lesion of or near the midbrain perhaps involving the internal geniculate bodies.

If, therefore, the audiometer curves show an abrupt loss in the high tones on both sides (above 2048 vibrations) and a tumor is known to be present, I think it may be a localizing sign of importance, i.e., to the region of the midbrain. Since cerebellar tumors, and tumors elsewhere, can cause the same curves, it can, of course, never be used for the determination of the location of the operative attack. The significance of the audiometer curve—if its import is later substantiated—would be much like that of destruction of the sella turcica, i.e., the effect might be from a direct or remote tumor.

In Case VIII there was practically total deafness on the side of the tumor, and in Case V, subtotal deafness in the contralateral ear. Since we have not been able to establish a central area for hearing, except in connection with the apparatus for speech, it has not seemed possible to place any significance upon such changes which would be strangely contradictory. Moreover, deafness without explainable cause is such a frequent occurrence, that one is not justified in regarding it as an effect of the tumor unless loss of vestibular function is also present. Our knowledge of the central anatomical pathways for hearing and its central representation is far from secure. That the usual textbook picture of a center for hearing in each temporal lobe is incorrect is at once proved by the complete extirpation of either temporal lobe, for subsequently there is not the slightest effect upon the audiometer curves.

However, in Case XII the audiometer curves (Fig. 60) show precisely the same outspoken bilateral loss of all the high tones that was recorded among the tumors in the third ventricle. The change is one of radical and not equivocal nature. And when the ventriculograms are inspected it will be seen that the

TABLE II

Tumors Presumably Benign That Have Been Found in the Literature

Case Reported By	Year Reported	Age	Sex	Duration of Symptoms	First Symptom	Intermittent Attacks	Headaches	Vomiting	Diplopia Extraocular Palsies	Dizziness	Bilateral Papilloedema	Hemiplegia or Hemianesthesia	Loss of Speech	Convulsions	Character of Convulsions	Staggering Gait	Mental Changes	Endocrine Changes	Other Signs or Symptoms	Tumor Disclosed at Necropsy	Tumor Disclosed at Operation	Clinical Diagnosis	Size of Tumor	Location of Tumor	Character of Tumor	Tumor Encapsulated	Gross Appearance	Microscopic	Hydrocephalus	Remarks
Henning and Wagner	1856	At birth			None															+			Large mass	Body of ventricle	Bone and cartilage	+	Large mass containing blood clots, cartilage and bone, surrounded by membrane	Bone, cartilage and connective tissue	+	
Broca	1861	25	F.																	+			25 grams	Right ventricle	Osteoma	+	Covered by membrane and attached by pedicle, not attached to choroid plexus	Bone with canaliculi	No	
Demange	1874	8			None															+		Child died in uremia during scarlet fever	Walnut	Left posterior horn	Ependymal glioma (?)	+	Attached to ventricular wall by pedicle	Like hyperplasia of deeper layers of ependyma	+	There were in addition numerous small excrescens from walls of both lateral and third ventricles
Cayley and Brown	1876	4 months		4 days				+	No					No					Localized bulge in parietal region over tumor; fontanelles tight	+			Duck's egg	Body of ventricle	Cyst	+	Cyst bulged through to surface of brain attached to dura			Sprang from choroid plexus. Clear serous fluid
Chambard	1881	Adult	M.																	+			70 grams	Left	Sarcoma ? or fibroma	+	Lobulated, round, attached to ependymal wall	Mostly connective tissue	+	Thinks it arose from choroid plexus. Calls it sarcoma, but its appearance is not unlike a fibroma
Audry	1886	45	M.	7 weeks	Headache		+					+					Elated and excited; later depressed		Coma; paralysis began in right *leg*	+			Walnut	Body left ventricle	?	+	Surface irregular; tumor is spongy and friable	Many large cells, all shapes, very compact, little connective tissue	+	Says it is like Guerard's case (probably malignant). Arose from the choroid plexus
Hirsch	1892	47	F.	1 month	Fainting attacks				No		None	+ Total	+	+	Jacksonian	+			Tenderness left frontal region	+			Walnut, 6 x 8 cm.	Body left ventricle	Spindle cell sarcoma (?)	+	Smooth, pale, red, contains cysts	Spindle cell sarcoma (?)	No	
Ströber	1893	1¼	M.	3 weeks	Vomiting			+	+					+					Bradycardia; rigid neck; fever	+		Tuberculous meningitis	Walnut	Left posterior horn	Choroid plexus	+	Nodular	Papillary structure of choroid plexus; some mucoid degeneration	+ On left	Choroid passes into tumor
Brüchanow	1898	2½	M.	2 years	Enlarging heart							+							Head large, sutures separated	+			Diameter 5 cm.	Left posterior horn		+	Nodular	Reproduces choroid plexus; many calcareous deposits	+	Thinks definitely arises from plexus
Collins	1899	20	F.	7 months	Headache		+										Progressive mental changes			+				Anterior horn, right ventricle	Calls it sarcoma	+				The absence of photographs and microscopic notes makes character of tumor uncertain
Hunziker	1905	27	F.	7 months	Headache left side		+	+		+	+	+ Face to hand to leg		+		+ To right	Depressed, forgetful; manner changed; unable to understand		Headaches like migraine; apraxia, astereognosis; tender left parietal	+			6 x 5 cm.	Left anterior horn	Ependymal glioma (?)	Not entirely	Hard greyish, red nodular, attached only to septum pellucidum, borders run into brain tissue	Round cells with sparse connective tissue, containing cysts	Only in both descending horns	Tumor may be glioma, or ependymal glioma
Borchers	1909	18	M.	5 months	Headache frontal		+ Frontal												Loss of vision	+			7 x 4 cm.	Body right ventricle		+				Arises from plexus
Jumon and Denet	1911	Child	F.																	+			Orange		Cyst					
Paulian and Aricesco	1930	30	F.	3 years	Headache		+ Worse in left parietal region		+			+ Facial							Lost vision; posterior clinoid processes destroyed	+				Left anterior horn		+	Pedunculated fibrous pedicle to wall of ventricle	Masses of cells of all sizes, no arrangement; calcospherites numerous		Patient died few hours after lumbar puncture
Jacarelli	1931	40	F.				+	+	No		+ Hemorrhages	Both legs worse left					Apathy; disorientation; memory poor; spinal fluid negative			+			44 grams	Right anterior horn		+	Soft, round			
Barre and Metzger	1928	26	M.	4 years	Headaches	+		+			+	+							Bilateral motor changes eventually; large calcified shadow	+						+	Calcified areas in tumor			Bilateral papilloedema only sign at first
Simon	1874	14	M.	Many years	Paralysis arms and legs												+		Patient is an idiot				Walnut	Posterior horn	Glioma	+	Hard, knotty and hard to cut	"Pinzel" cell glioma	+ Marked	
Davis and Cushing (Case II)	1925	9	M.	10 months	Headaches		General at first; later frontal	+		+	+	No		+					Enlargement of head; blind 6 months; rigid neck; anosmia; nystagmus	+	No	Cerebellar tumor	Hen's egg	Left ventricle posterior part		+	Hard tumor surrounded by cystic cavities, lined by smooth membranes	Like choroid plexus	+ Marked	Cerebellar operation performed
Davis and Cushing (Case III)	1925	50	M.	6 months	Writing and speech affected	No	No				No	Partial	Affected	+	Jacksonian				Paraphasia; difficulty writing		+	Cerebral tumor	Large cyst	Parietal lobe	Cyst with nodule in wall	Nodule in wall	Cyst with mural nodule	Like choroid plexus		Face, arm, leg involved in this order. Small piece of tumor removed for microscopic study. Died year later. No autopsy
Wätzold	1905	4½	F.	9 months																+			160 grams		Sarcoma ?	+	Knotty surface	Perithelial structure		
Claude and Loyez	1913				Hemiplegia															+			2 x 1.25 cm.	Anterior horn	Hard	+	Attached to ventricular wall by small pedicle; slightly irregular nodular surface	Neuroglial elements but covered by ependyma		Author thinks it is like granular ependymitis
Lydston	1915	21	M.	5 years	Headaches	+	+												Loss of vision; bulging head over tumor	+			Large cyst	Anterior horn	Cyst with slightly yellow fluid	Cyst	Large single cyst, smooth wall; no solid tumor seen	Not made	High grade	One cannot believe that a solid tumor of small size is not in the wall of cyst
Jumentié, Olivier and Leclaire	1924, also 1927	35	M.	3 years	Headache		+	+		+	+	No		+	Unilateral (left)	+	+ Irritable, forgetful, timid and defiant		Loss of sphincter control; lumbar puncture —pressure increased	+		Brain tumor		Right	Perhaps an ependymal glioma		No invasion of depth of brain	Glioma		Treated by radium. Attacks improved
Hart	1910	50	F.				+				+	+		+			Loss of memory			+			Goose egg	Right			Imbedded in brain	Like choroid plexus		A second smaller tumor is in the other lateral ventricle
Van Wagenen	1932	3 months	F.	1 month	Bilateral squint; enlargement of head	Too young	Too young	+	+			No		No							+	Brain tumor		Right posterior and descending horns	Adenoma of choroid plexus	+	Encapsulated tumor in posterior horn of right ventricle	Like choroid plexus	+ In descending horn of right ventricle	Tumor was removed in fragments with the cautery. Lived 2½ years. Died of other causes. Necropsy obtained. No recurrence

posterior half of the third ventricle is obliterated (Fig. 61C). Its effects, therefore, are upon the midbrain. At the time the audiometer curves were seen it was my belief that the patient probably had a tumor of the pineal, third ventricle or environs.

X-RAYS OF THE HEAD

X-rays of the head—even with the greatly improved qualities of recent years—have been most disappointing in defining the location of these tumors. The only pathognomonic finding would, of course, be an area of calcification in the tumor. In not a single case of our series was this calcification present. However, Barré and Metzger reported a large calcified shadow—the only one in the literature—in their tumor.

It will be recalled that calcification of the glomus of the choroid plexus of one or both sides is a not uncommon normal finding. One must weigh this possibility in the balance before making a positive diagnosis of a tumor from such a shadow. The normal choroidal calcifications occur in well established positions and are usually but not always bilateral and symmetrical. Together with a calcified pineal shadow which is also usually present, a triangle with equal sides is formed. By deformation of this triangle it should be possible to diagnose and localize an occasional brain tumor, just as with the shift of the pineal shadows, but as yet this diagnostic possibility has been of little importance.

That definite changes due to the tumor may be present in the roentgenograms is evident from the accompanying table. They are, however, alterations in the skull due either to local or general pressure, or to both. In no instance are any of these changes of value in localizing the tumor to the point necessary for operative attack. They may in fact be most misleading if emphasized unduly.

In twelve of our fifteen cases the x-rays—which have been studied with care—have been negative in every respect. In three cases there has been generalized convolutional atrophy which only indicates intracranial pressure, but is usually due to

hydrocephalus. In Case III, a child of 12 years, the frontoparietal and parietal-occipital sutures were separated. Since on the law of probabilities, convolutional atrophy and separation of sutures are usually due to tumors in the posterior fossa, this finding may well lead, as it has so frequently done, to misplaced subtentorial operations.

Destruction of the posterior clinoid process is reported by Paulian and Aricesco. Absence of the sellar landmarks by no means denotes a hypophyseal tumor, as is so frequently assumed. Usually the tumor is nearby, but it may be in any direction—even in the posterior cranial fossa; the tumor may not actually be in contact with the sella. It is, therefore, needless to add that destruction of the sella without the all-important accompanying hemianopsia is by no means indication for operating upon the hypophyseal region.

LUMBAR PUNCTURE IN DIAGNOSIS

Nothing whatever is to be gained, either in diagnosis or localization, by a lumbar puncture. The existence of intracranial pressure, which in these cases is the only evidence of value in a lumber puncture, is perfectly well known by the patient's signs and symptoms. The great risk attending lumbar punctures in the presence of intracranial pressure should deter one from their use when nothing is to be gained in return. It is worthy of note that Paulian and Aricesco's patient died a few hours following a lumbar puncture.

CAN A DIAGNOSIS OF TUMOR IN THE LATERAL VENTRICLE BE MADE BY NEUROLOGICAL FINDINGS AND THE X-RAY? IS THERE A CLINICAL SYNDROME?

From our series of cases and those from the literature (40 in all) in not a single instance was the preoperative diagnosis of a tumor in the lateral ventricle made from the neurological findings. And with the x-ray a diagnosis was possible only when there chanced to be a large calcified shadow. It was hoped that from a careful analysis of the subjective

and objective findings it might be possible to look back and improve on the results of the future. I cannot find a clinical syndrome that will serve this purpose. It is quite true that in those cases with hemiplegia of rapid onset one may think of the possibility of a tumor in the lateral ventricle, and particularly when motor changes appear on the other side of the body in addition, but precisely these findings are obtained and with even greater frequency in other brain tumors. The periodicity of attacks of intracranial pressure without localizing signs also makes one think of a ventricular tumor acting as a ball-valve; but a tumor in the third or fourth ventricle is just as probable as one in the lateral ventricle. If, in addition to the intermittent attacks of intracranial pressure, there is transient hemiplegia, one may, with more reason, suspect a tumor in the lateral ventricle; but other cerebral tumors produce exactly the same symptoms and with much greater frequency. At best one can do little more than suspect a tumor in the lateral ventricle. Certainly it would never be justifiable to operate without far greater security in localization. The results would scarcely be better in the future than in the past. I must confess a very serious doubt that a clinical syndrome for tumors in the lateral ventricles can ever be established.

CHAPTER VI

LOCALIZATION OF TUMORS IN THE LATERAL VENTRICLE BY VENTRICULOGRAPHY

The use of ventriculography has made it possible to overcome all of the diagnostic defects in this group of tumors, indeed of all intracranial tumors producing pressure and many that do not cause pressure. When correctly used and interpreted, all tumors in the lateral ventricle (causing pressure) can be localized with absolute precision. If used correctly, there is no attendant risk. I refer only to the introduction of air into the lateral ventricles. The injection of air into the spinal cord is attended by very grave risks, and is most capricious in its results. In the presence of all tumors or suspected tumors only the direct ventricular injections should be used. It is essential that the air be removed immediately after the x-rays have been made and interpreted; and it is equally essential that the tumor be attacked and removed immediately thereafter in order that the remaining air may not be retained in the ventricular system behind the obstructing tumor where it cannot be absorbed. If the tumor is removed any remaining air passes freely to the subarachnoid space where it is quickly absorbed.

From the series of fifteen tumors localization was made by ventriculography in eleven, by ventricular estimation in two, by neurological signs in one and by a misdirected cerebellar operation in one.

The changes induced in the ventricular system by tumors in the lateral ventricles are (*a*) border or filling defect of the tumor, (*b*) hydrocephalus, (*c*) deformation and dislocation of the ventricular system.

a. BORDER OR FILLING DEFECT OF THE TUMOR

Every tumor blocking the lateral ventricle will show a sharp border which will be the terminus of the air shadow, i.e., the

line of obstruction. Whether the anterior or posterior border of the tumor is shown in the ventriculogram depends upon which ventricle is injected (if the injection is made at the customary site over the posterior horn). If the ventricle on the side of the tumor is injected the posterior border of the tumor will show (Case II); if the injection is made into the contralateral ventricle, the anterior border of the tumor will appear (Cases I, II, XI and XII). Occasionally, as in Case II, it may be advisable or necessary to inject both ventricles; since both the anterior and posterior borders of the tumor are then defined, the tumor must occupy the space between. With such findings the exact size of the tumor will be known before operation. In most ventricular tumors the posterior horn will be obliterated on the side of the neoplasm. At times there is a filling defect in the ventricular shadow caused by the tumor bulging into the ventricle. Many small tumors, particularly those of the glomus of the choroid plexus, cause such filling defects but they do not cause symptoms because the air passes freely around them. On the other hand, among the symptom-producing tumors a filling defect is not uncommon, but for this to be present there must be in addition an obstruction in the ventricular system; this is nearly always at the foramen of Monro, and is the real source of the intracranial pressure. Filling defects of this character are shown in the ventriculograms of Cases V, VI, VIII and IX. In such cases disclosure of the filling defect is of the utmost importance in differentiating tumors in the lateral ventricle from those in the third ventricle. This information is absolutely essential in determining the side of the head on which the operative exposure is to be made. It is evident, therefore, that all filling defects of symptom-producing *primary* tumors in the lateral ventricle will be on the mesial side of the lateral ventricle; otherwise the foramen of Monro would not be blocked. A filling defect in the outer wall of the lateral ventricle in a case of intracranial pressure is usually due to an intracerebral glioma that is bursting into and partially filling the ventricle.

In these cases the lateral ventricle will rarely attain the great size that results when the foramen of Monro is blocked by a primary tumor in the third or lateral ventricle (there are exceptions).

b. HYDROCEPHALUS

Hydrocephalus results only when the tumor blocks the ventricular system at or posterior to the foramen of Monro, but in only one case (VII) from the series was hydrocephalus absent. Hydrocephalus results in two ways and assumes two different expressions, which if not properly interpreted, may lead to mistaken localization. When the tumor obstructs a lateral ventricle, localized hydrocephalus will always result in the isolated part of the ventricle (Cases II, XI and XV). Or if the foramen of Monro is obstructed the entire lateral ventricle of that side will become hydrocephalic.

Such localized hydrocephalus may perhaps be called primary in contrast to another or secondary form which develops, at times only. This additional manifestation of hydrocephalus is caused by the lateral compression of the aqueduct of Sylvius or third ventricle. When this occurs hydrocephalus results in the lateral ventricle opposite the tumor, the third ventricle in part or whole, depending upon the point of obstruction, and at times, but not always, the anterior portion of the lateral ventricle on the side of the tumor; the exact result of the latter effect is dependent upon how much this part of the ventricle is reduced by the tumor's bulk. The secondary hydrocephalus, so-called, is precisely similar to that resulting in nearly all large tumors in the posterior half of the cerebral hemispheres and necessarily compressing the iter or third ventricle, or both. Case XII is an example of the secondary hydrocephalus so resulting. Air was injected into the left ventricle, i.e., opposite the tumor; there was marked hydrocephalus of this ventricle and the anterior half of the third ventricle. The right ventricle was small and showed a sharp oblique border well anteriorly. The third ventricle showed a circular filling defect

where it was obstructed by the lateral (secondary) compression of the tumor. It was, of course, not necessary to inject the descending horn of the right lateral ventricle, but the existence of a localized hydrocephalus in this obstructed part of the ventricle is inescapable. If only superficially examined, one might suppose that, in this case, the tumor was in the third ventricle, but if this were true the right foramen of Monro and the right lateral ventricle would be as large as the left. Moreover, the third ventricle was bent slightly towards the left side. We know that unimpeded passage of air into an enlarged right lateral ventricle would result from an obstruction of only the posterior half of the third ventricle. Since this is not true there must be a tumor on the right side; and its anterior border must be represented by the posterior margin of the air shadow in the anterior horn.

In Case XI a tumor of the same kind, in exactly the same position, and of even larger size, caused only a localized hydrocephalus in the descending horn. The remainder of the ventricular system was quite small. Doubtless the explanation of the failure of the iter or third ventricle to be obstructed was that the tumor arose in early life when the skull enlarged locally and permitted the pressure to be directed from these channels.

c. DISLOCATION AND DEFORMATION OF THE VENTRICULAR SYSTEM

In all intraventricular tumors producing pressure there are dislocations and deformations of parts of the ventricular system. Dislocations are best disclosed in the anteroposterior views of the ventriculograms. When tumors are located in the posterior part of the cerebral hemispheres the amount of dislocation may be relatively less, but it is always present and will be evident by some obliquity of the third ventricle. Deformations and absence of localized parts of the lateral ventricle will also be evident. In only one case (VII) of the series was the tumor so situated—in the anterior horn—that hydro-

cephalus was entirely absent. The ventriculographic changes were, therefore, precisely like those of any other encapsulated tumor in the frontal region, i.e., dislocation of the entire ventricular system and deformation of the lateral ventricle in the neighborhood of the tumor.

LOCALIZATION BY VENTRICULAR ESTIMATION

When, in preparation for ventriculography, a cyst or hematoma is tapped, the localization of the tumor is, of course, absolute. In Case X a cyst, and in Case XV a hematoma, were encountered. VanWagenen's successful case was also localized when a cyst was encountered by a ventricular tap.

CHAPTER VII

OPERATIVE TREATMENT OF VENTRICULAR TUMORS

Needless to say there is no medical therapy and no treatment by x-rays of any kind that can produce the slightest benefit to patients afflicted with these tumors. Nor is there any indication for, or possible benefit in, palliative operative efforts by decompression. The only treatment is complete removal of the tumor and this should be done as soon as the diagnosis is established. Since the intracranial pressure advances with great rapidity after occlusion of any part of the ventricular system, delay in operative treatment of these, as indeed with all brain tumors, may be responsible for the patient's death.

Absolute precision in localization of the tumor—by ventriculography—must dictate the exact site of the operative exposure. A misplaced exposure is certain to be productive of extensive injury to the brain and will probably end in a fatality.

In order to expose the tumor an area of cerebral cortex of varying size and overlying the tumor must be excised. At times even a large part of a frontal lobe of either side may be removed for this purpose. If, as sometimes obtains, the overlying cortex is a functioning part of the brain, the cortical defect must be made to one side and the tumor attacked through this opening as a tunnel. It is doubtless possible to remove an occasional avascular tumor through a simple incision through the cortex, but there is nothing to be gained by such a restricted opening, and should bleeding occur at such great depth, it would be exceedingly difficult and probably impossible to control. It is far more important to provide an adequate approach through which any operative difficulties may be at once under complete control. An enlarged lateral ventricle is always an asset in attacking tumors within the

ventricle. Release of the contained fluid reduces the increased pressure and, therefore, the cerebral protrusion. Indeed a preoperative knowledge of the size of the ventricles is most important in determining the size of the projected bone flap. The smaller the bone flap the better, but it must be large enough to allow any cerebral protrusion (from pressure) to be properly accommodated without rupture. If the ventricle is quite large a small bone flap will be ample; if there is little or no enlargement of the ventricles a large bone flap will be required.

The method of attack upon the tumor must vary with its character. A small tumor in a large ventricle (as in Cases I, V, VI, IX and XV) can be dissected out slowly and with painstaking care. When possible, removal of the interior of a larger tumor with a curette, and finally removal of the capsule by careful dissection, as in Case VIII should be carried out. Finally in the large and very hard tumors such as Cases VII, XI and XII, XIII and XIV the removal is only possible by enucleation with the index finger. When the extirpation can be done in a painstaking fashion, hemorrhage is usually readily controlled by the appliction of the cautery to the bleeding points as they occur. But when it is necessary to resort to rapid enucleation with the finger, a desperate battle with hemorrhage will probably ensue, for the large, hard tumors are usually exceedingly vascular. Particularly is this true of the huge tumors (such as Cases XI, XII, XIII and XIV) arising in the glomus of the choroid plexus, and from which the tumor gets its blood supply. The sudden tear of the great tortuous arteries and veins passing from the glomus of the plexus releases a furious hemorrhage which must be quickly packed with wet cotton; later, as the cotton is gradually elevated, the broken vessels are isolated, picked up with forceps and sealed by application of the electro-cautery.

The danger attending the removal of intraventricular tumors is due to (1) postoperative cerebral oedema, or (2) hemorrhage which may occur either at operation or subsequently. Post-

operative oedema occurs during a period of several days following the operation. It is dependent upon the degree of trauma inflicted upon the brain at the time of operation and can be fairly well estimated at that time. If the ventricular dilatation is marked, subsequent tappings will amply take care of the pressure. If the ventricles are small and pressure remains at the end of the operation, a decompression or removal of a small bone flap may be necessary. Lumbar punctures do not help and may be quite harmful.

To combat the effects of severe hemorrhage at the time of operation intravenous injections of glucose or blood are effective. I am quite certain that Case XII was saved only by an immediate transfusion of blood, in anticipation of which the patient was matched before operation. Postoperative bleeding, formerly so common, is now fortunately quite rare. More effective hemostasis is now afforded by the electro-cautery, but of greater importance is the absence of venous enlargement due to postoperative vomiting since the introduction of avertin anesthesia. It was from the latter complication that our two deaths (Cases IV and IX) occurred. The third death in the series followed a misplaced cerebellar operative exposure. It need hardly be added that a fatality is an almost necessary sequel to any misplaced cranial exposure.

ULTIMATE RESULTS TO LIFE AND FUNCTION

The operative mortality in the series has been 20 per cent—a percentage that is now much reduced with present technical improvements in cerebral surgery and with our better understanding of the nature of these tumors. In the last seven cases of the series the tumors have been totally enucleated without a death.

In the twelve living patients there has, as yet, been no sign of recurrence of the tumor. But it will still require much additional time to deny this possibility. The longest survival period is fifteen years (Case I), the next, thirteen years (Case II). Since the tumors were so well encapsulated that they

apparently could be removed entirely, and since they are seemingly entirely different from the intracerebral tumors that do recur, I am tempted to believe that recurrence will be unlikely.

A severe degree of motor weakness persists in Case II, and a much lesser degree in Case XV, but both were hemiplegic at the time of operation. A moderate degree of motor weakness persists in Cases VIII and XIV. In none of the remaining cases is there any motor dysfunction. In Case XII slight weakness of the arm followed immediately after the operation but quickly cleared.

Alexia, agraphia and anomia resulted after the operation in Case XI, in which it was necessary to remove the left supramarginal and angular gyri to approach the tumor. These functions have in large part returned. Homonymous hemianopsia persists in Cases XI, XII, XIII, XIV and XV. Subsequent convulsions of Jacksonian or focal character have occurred occasionally in six of the twelve surviving patients. The incidence may eventually be somewhat higher for several cases have been operated upon only recently and the usual story is that convulsions begin between one and two years after the removal of the tumor, or for that matter after any severe cranial trauma. In Case I there were occasional convulsions for the first ten years and none during the past five years. It is perhaps worthy of note that two of the six patients who have had convulsions subsequent to the operation had them before operation; and one patient who had them before has had none since, although six years have since elapsed. As a rule convulsions that persist following the removal of cerebral tumors are never frequent and gradually diminish over a long period of time. The incidence and subsequent course of convulsions following the enucleation of intraventricular tumors is perhaps somewhat higher than in cases of the removal of other tumors in the cerebral hemispheres. The cause of such convulsions, of course, is the cerebral defect.

CHAPTER VIII

PATHOLOGY

Of several tumors in the series there can be no doubt whatever concerning their character. Four of these (Cases X, XI, XII and XIII) are known to have arisen in the glomus of the choroid plexus—the only ones in which this origin is clearly established by the gross attachments. Case XV is a non-indigenous angioma that might have arisen anywhere in the brain, or elsewhere in the body. In Case X the tumor is clearly an adenoma of the choroid plexus; the characteristic adult epithelium of the choroid is more or less perfectly reproduced, together, in many places, with its alveolar structure. The difficulty of differentiating the benign adenomatous forms of choroid plexus epithelial tumors from the malignant type, which is much more common, has been emphasized in the discussion of those known to be malignant. The tumors in Cases XI, XII and XIII are pure fibromata of the choroid plexus and are not greatly different from fibromata arising elsewhere in the brain or other parts of the body. These three tumors are almost identical in every gross and microscopic respect and, therefore, doubtless represent a distinct and not uncommon type. The histological appearance of these tumors is perhaps not greatly different from patches of fibrous tissue that are seen in some of the small symptomless tumors of the plexus. Since the specimens in Cases III and VI were lost (after they were drawn) the microscopic notes are lacking.

The classification of the remaining tumors cannot be made so unequivocally. All are, I believe, different from other intracranial tumors, excepting some from the third ventricle. Possibly the fourth ventricle may harbor similar growths, but if so I have not seen them. The fact that these tumors are so different suggests their origin from either of the two structures in

the ventricle that are different from the remainder of the brain, namely, the ependyma and the choroid plexus. This suggestion appears to be the more probable because some of the tumors in the third ventricle are quite similar. But if this is true, one wonders why similar tumors do not arise in the ependymal lining of the fourth ventricle or from the choroid plexus in the posterior cranial fossa. A partial answer to this query may be that the other benign symptomless tumors in the choroid plexus of the lateral ventricle are rarely, if ever, seen in the choroid plexus of the posterior fossa. For some unknown reason the cysts, psammomata, and fibromata are common in the former and exceedingly rare in the latter. These tumors are at once stamped in the gross by their encapsulation, or near-encapsulation, a feature that never occurs with other tumors arising from the primary cerebral tissues. When encapsulation is not absolutely complete, there is only a very superficial attachment to, and never an invasion of, the ependyma. It is these two characteristics of benignity, correlated with certain clinical proof of cures (in a number of cases) over a period of several years that leads us to classify them as benign tumors.

Two tumors in the series were very much alike in the gross (VIII and IX) and quite different from the remaining tumors of the lateral ventricle, but they are quite dissimilar under the microscope. They were soft, fluffy, papillomatous tumors that would waft about in the ventricle like seaweed and were superficially attached by a broad base to the wall of the ventricle. One might well have anticipated an epithelial papillomatous structure in each. In the former (Case VIII) small round cells are uniform throughout the sections—a picture much like that of Case IV which has an entirely different gross structure. In the latter (Case IX) remarkable large cells predominate.

The histological structure of the tumor in Case IX is particularly interesting. The masses of large cells of variable size and shape with vesicular and excentrically placed nuclei

are unlike any other tumor in this series, but are, I think, identical with two tumors of the third ventricle recently reported (Cases VI and VII of Group II).[1] But here again the gross appearance of this tumor has no resemblance whatever to those in the third ventricle (one really had its point of origin in the septum pellucidum and projected into the third ventricle). Both of the latter tumors were hard and encapsulated, the former soft and papillomatous. In none of the three tumors has there been any reason to think there might be metastases. One patient is well two years after removal of the tumor. Examination of the brains post-mortem in the other two cases (autopsies were restricted to the head) revealed no other nodules.

Dr. Arnold Rich, to whom I have referred these and other tumors of the series for expert opinion, has not seen cells of this character in other tumors of the brain or elsewhere. Since they are unlike adult cells lining the ventricles or choroid plexus, they must represent their cellular antecedents and be classified among the tumors arising from embryonal cells. The tendency to syncytial masses and giant cells perhaps also supports such an assumption. The excentrically placed nuclei, so similar in each specimen, suggests but does not prove a relationship to the adult ependymal cells which are so characterized. Their ventricular attachment being at a distance from the choroid plexus in two cases would lend support to the impression that the cells are of ependymal and not of plexus origin. It is worthy of note that occasional strands of glial fibers are visible in the sections.

The remaining tumors—I, II, IV, V, VII and XIV—(III and VI have no microscopic examinations) would appear to fall more or less together. When the cytological composion of these tumors is matched with the gross appearance, one must confess a feeling of surprise to find, not an epithelial but a fibrous structure. This would not be so disturbing in the

[1] Benign Tumors in the Third Ventricle of the Brain; Diagnosis and Treatment. Published by Charles C. Thomas, 1933.

choroid plexus where fibrous tissue might well be expected, but we are accustomed to consider the subepithelial layer of ependyma as a type of glial tissue that would have the same general character as glia elsewhere in the brain. This surprise is perhaps lessened when glial stains show only trivial amounts of glial fibers, so scant indeed that one must frequently search over several fields before finding traces of them. On the other hand, glial tissue made up the bulk of the tumor in one of these cases (V)—the only one of the series—and still it was one of the most perfectly encapsulated tumors of the group. Similar encapsulated tumors with abundance of glial tissue were present among the series of neoplasms in the third ventricle.

The tumor in Case VII is so stony-hard and nodular that it would readily pass for a dural endothelioma. It is made up of fibrous tissues of varying texture. This tumor is known not to have arisen from the choroid plexus, because it filled the anterior horn which does not contain choroid plexus. I think it safe to conclude that the connective tissue must have arisen from the subepithelial layer of ependyma. Occasional tiny patches of glial fibers are found in the sections.

The tumor in Case II was nearly as hard, but entirely confined to the ventricle. It, too, is made up of fibrous tissue of looser texture and with occasional strands of glia. Cases IV, V and XIV were also hard, nodular growths, not greatly dissimilar in gross appearance, but there is apparently little in common in the microscopic cellular appearance. In the former, one might perhaps, if the examination were hastily made, make a diagnosis of small round cell sarcoma, or possibly, if it was known to arise in the brain, a cellular glioma; and in Cases V and XIV the diagnosis of a fibroma would be in order until the abundance of glia would demand consideration of the diagnosis of glioma.

The tumor in Case I was soft, reddish-brown, and had burrowed through the brain reaching the surface. Except for its encapsulation and unquestioned origin it would not appear

greatly different from the cerebral gliomata. But the tumor is also one of the fibrous growths.

Perhaps, therefore, a diagnosis of ependymal fibromata or glioma may be made tentatively in Cases I, II, V, VII and XIV. It is difficult to suggest even a tentative diagnosis in Cases IV and VIII, which look quite unlike all the others under the microscope. They are far too cellular to be grouped among fibromata and primary sarcoma of the brain, which might be suggested, is almost unknown. Moreover, a survival period of five years since the operation in Case VIII would certainly establish it as a benign tumor; and this patient had symptoms for nine years before the operation, much too long for a malignant tumor. Such are the hazards attending an unsupported histological diagnosis of tumors yet to be classified.

It is perhaps worthy of note in passing that none of the striking "colloid cysts" in the third ventricle have been found among the tumors in the lateral ventricle. I had come to the conclusion, from the evidence at hand, that they arose from congenital enlage, perhaps referable to the ependymal epithelium. I have since found one in the stalk of the hypophysis, but none elsewhere.

The seeming conflicts between the gross and microscopic appearances of this group of tumors cannot be harmonized by the writer. I am forced to accept the gross evidence that the tumors are benign, to place many of them as ependymal tumors, though with misgivings, and defer the ultimate microscopic classification to more competent students of pathology.

CHAPTER IX

SUMMARY AND CONCLUSIONS

1. A series of fifteen cases of primary benign encapsulated tumors in the lateral ventricles is reported. All have been found at operation and all but one totally removed. Twelve patients recovered and three died. There has been no death in the last seven cases and but one death in the last eleven cases.

2. Twenty-five additional cases have been collected from the literature. With two exceptions these tumors have been disclosed after death.

3. The clinical signs and symptoms have been analyzed in all cases. The most common signs and symptoms are: headache, vomiting, dizziness, papilloedema, diplopia with or without extra-ocular palsies, focal or Jacksonian convulsions, hemiplegia and corresponding reflex changes, and hemianesthesia; but there is no clinical syndrome by which these tumors can be localized.

4. Perhaps the most suggestive symptom of a ventricular tumor is intermittent headaches, but these are equally suggestive of tumors acting like a ball-valve in any other part of the ventricular system.

5. By ventriculography all tumors in the lateral ventricle can be accurately diagnosed and localized with precision. This procedure when used correctly is without any risk to life or function.

6. In some cases the tumors, cysts, or hematomata are encountered when the ventricular puncture is made (ventricular estimation).

7. Every brain tumor causing signs or symptoms of intracranial pressure will change the size, shape, or position of some part of the ventricular shadows. All of the intraventricular

tumors of this group caused symptoms by occluding the lateral ventricle. Always the line of the ventricular obstruction will be the border of the tumor. All pressure-producing ventricular tumors, therefore, cause hydrocephalus in the occluded part of the lateral ventricle (localized hydrocephalus) and many also obliterate the aqueduct of Sylvian or third ventricle, or both, and cause hydrocephalus in the remainder of the obstructed ventricular system.

8. To approach the tumor preparatory to operative removal, it is necessary to uncover the growth through a transcortical incision of adequate length, or by removing an area of cortex directly over or adjacent to the tumor. Important functioning areas of the brain must be avoided when sacrificing the cerebral cortex for exposure.

9. Since all of the tumors are encapsulated, they are curable when completely extirpated. This is, therefore, the only satisfactory treatment.

10. The risks attending the removal of these deep seated tumors are now greatly reduced by the use of the electrocautery, continuous suction, and avertin anesthesia.

11. The intraventricular tumors are of varying kinds—ependymal fibroma or glioma, adenoma and fibroma of the choroid plexus, embryonal ependymal tumor, angioma. They arise from the ependyma, subependymal layer, and the component parts of the choroid plexus. Many arise from the glomus of the choroid plexus.

12. A number of small, symptomless, and malignant tumors, principally of the choroid plexus, are also recorded. It is thought to be possible, but is beyond proof, that these tumors may be the forerunners of the group of larger tumors that cause symptoms. The malignant tumors of the choroid plexus—so-called adenocarcinoma—are rather difficult to differentiate histologically from those of benign type. They have been reported in far greater numbers than the benign. They metastasize to the brain and elsewhere in the body. They recur when removed, are invasive, and deposit seeds which are dis-

seminated through the cerebrospinal fluid producing numerous secondary nodules.

13. The small primary symptomless tumors occur frequently and are of much the same types as those producing symptoms. Several of these tumors have been removed at operations for hydrocephalus. They may be bilateral. Cysts and psammomata of the choroid plexus are additional tumors of smaller type that occur with even greater frequency.

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